ASSOCIATION BETWEEN SERUM TESTOSTERONE CONCENTRATION AND CORONARY HEART DISEASE

Dissertation
Submitted in partial fulfillment of the regulations of

M.D. DEGREE EXAMINATION

BRANCH I GENERAL MEDICINE

Department of General Medicine

GOVT. STANLEY MEDICAL COLLEGE AND HOSPITAL

CHENNAI - 600001

THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY

CHENNAI

APRIL 2014

CERTIFICATE

This is to certify that this dissertation titled

"ASSOCIATION BETWEEN SERUM TESTOSTERONE CONCENTRATION AND CORONARY HEART DISEASE"

is the bonafide work done by **Dr. Sathesh Kumar. K.,** Post Graduate student (2011 – 2014) in the Department of General Medicine, Government Stanley Medical College and Hospital, Chennai under my direct guidance and supervision, in partial fulfillment of the regulations of The Tamil Nadu Dr. M.G.R Medical University, Chennai for the award of M.D., Degree (General Medicine) Branch - I, Examination to be held in April 2014.

Prof.Dr.G.VASUMATHI, M.D.,
Professor of Medicine,
Dept. of General Medicine,
Stanley Medical College,
Chennai-600001.

Prof.Dr.P.VIJAYARAGAVAN, M.D.,
Professor and Head of Medicine,
Dept. of General Medicine,
Stanley Medical College,
Chennai-600001.

PROF. S. GEETHA LAKSHMI, M.D., PhD,
The Dean**,**
Stanley Medical College,
Chennai-600001.

DECLARATION

I, **DR. K. SATHESH KUMAR** solemnly declare that this dissertation titled "**ASSOCIATION BETWEEN SERUM TESTOSTERONE CONCENTRATION AND CORONARY HEART DISEASE**" is a bonafide work done by me in the Department of General Surgery, Government Stanley Medical College and Hospital, Chennai under the guidance and supervision of my unit chief.

Prof.Dr.G.VASUMATHI, M.D.,
Professor of Medicine

This dissertation is submitted to The Tamilnadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the university regulations for the award of M.D., Degree (General Medicine) Branch - I, Examination to be held in April 2014.

Place: Chennai.
Date: December 2013. **DR. K. SATHESH KUMAR**

ACKNOWLEDGEMENT

I am grateful to **Prof. Dr. S. Geethalakshmi,** Dean, Govt. Stanley Medical College for permitting me to conduct the study and use the resources of the College.

My sincere thanks to **Prof. Dr.P.Vijayaragavan, M.D.,** Professor and HOD, Department of General Surgery, for his valuable guidance throughout the study.

I am highly indebted to my guide **Prof.Dr.G.Vasumathi, M.D.,** Professor of Surgery for her constant help, inspiration and valuable advice in preparing this dissertation.

I express my deepest sense of thankfulness to my Assistant Professors **Dr.Ramalingam,M.D.**, **Dr.Sujatha,M.D.,**, **Dr. Geetha,M.D., Dr. Raja Kumar,M.D.,** for their valuable inputs and constant encouragement without which this dissertation could not have been completed.

I am particularly thankful to my fellow Super Specialty Post Graduate Dr. Azimudin Haja for his valuable support in the time of need throughout this study.

I would be failing in my duty without acknowledging the contribution of Dr.Priyavadhana, Dr. Rajkamal in helping me in completing this dissertation

It is my earnest duty to thank **my wife Mrs.S.Pavithra** without whom accomplishing this task would have been impossible.

I am extremely thankful to my patients who consented and participated to make this study possible.

Overall it is the blessing of **my father Mr.N.Kanagasabapathy** without which accomplishing this task would have been impossible.

TABLE OF CONTENTS

INFORMATION FORM

TURNTIN SCREEN SHOT

MASTER CHART

INTRODUCTION

Acute coronary syndrome includes unstable angina and non-ST elevated myocardial infarction and is defined as a spectrum of disease characterized by either:

1. New onset angina.
2. Angina at rest
3. Progression of angina of increasing frequency or severity.
4. Angina in response to lower levels of exertion.
5. STEMI

Acute coronary syndrome most often represents acute atherosclerotic plaque rupture with exposure of thrombogenic sub-endothelial matrix.

Coronary atheroma occurs as a result of inflammatory process. Cellular inflammation and local inflammation in the arterial wall occurs as a result of cytokines, which can further progress to cause vascular smooth muscle apoptosis, degradation of the fibrin cap and plaque rupture.

Clinically patient can have unstable angina or progressed to myocardial infarction as a consequence of thrombus formation due to platelet aggregation. Cytokines have pivotal role in pathogenesis of atheroma formation. Pro-inflammatory cytokines are suppressed by testosterone which shows immune-modulating effect. Men with low testosterone levels are at increase risk of developing acute coronary syndrome.

An anti-inflammatory effect of normal physiological levels of sex hormones may therefore, be important in atheroma protection. Cytokines and C-reactive proteins are found to be elevated in coronary heart disease patients.

Low serum testosterone levels is found in patients with coronary heart disease. Two most important activities in the pathogenesis of atheroma formation are cytokine activation and vascular smooth muscle cell apoptosis.

Main action of testosterone hormone is to deactivate cytokines as a result of which smooth muscle apoptosis is inhibited and leads to smooth muscle proliferation. So, as a consequence of this smooth muscle proliferation action by this hormone, it helps in maintaining the fibrous cap of the atherosclerotic plaque.

Testosterone hormone has calcium channel antagonism which results in coronary artery vasodilation. Various studies have proved the beneficial effect of testosterone hormone in coronary heart disease patients.

The following study is done to show the association of serum testosterone levels in patients with coronary heart disease.

AIMS AND OBJECTIVES

- To estimate the level of free testosterone level in male patient suffering acute coronary syndrome.

- To correlate free testosterone level with mortality and morbidity in male patients presenting with acute coronary syndrome.

- To correlate free testosterone level in male patients of acute coronary syndrome with other parameters e.g. BMI, lipid abnormality & ECG changes.

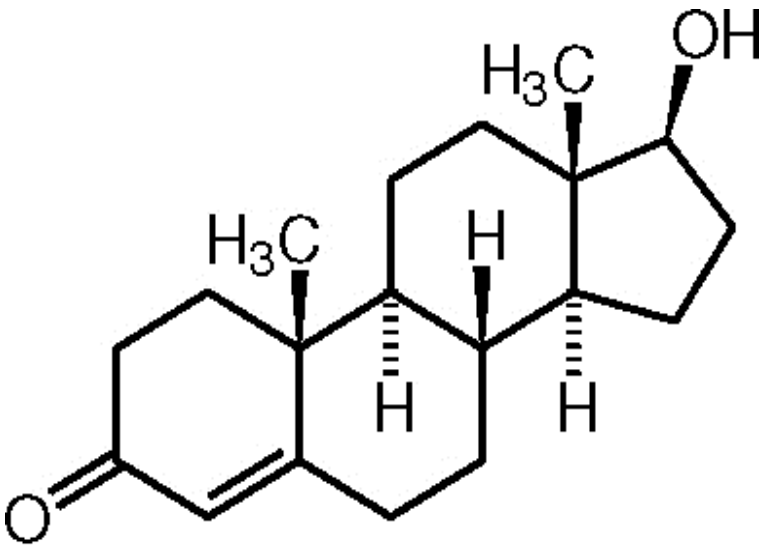
REVIEW OF LITERATURE

Atherosclerotic coronary artery disease (CAD) is a leading cause of mortality and morbidity all over the world. Men are more than twice as likely to develop CAD. Incidence of coronary heart disease in females when compared to male is low before menopause but it is of same incidence after menopause.

TESTOSTERONE

Leydig cells of testis produces testosterone which is synthesized from cholesterol and also from androstenidione secreted by the adrenal cortex. Leydig cells which is present in testis is controlled by LH for the secretion of hormone testosterone. Normal level of testosterone in adult – 4 to9ng/day secretion. Testosterone bound to protein is 98% and only 2% is free. Out of that 985 bound form65% is combined with beta globulin called gonadal steroid binding globulin and 33% bound to albumin.

Minimal quantity of testosterone is converted to estradiol but maximum quantity is converted to 17-keto steroids (like androsterone and etiocholanolone) and excreted in urine. About 70% of urinary 17-keto steroids are of adrenal origin and 30% of testicular origin.



testosterone

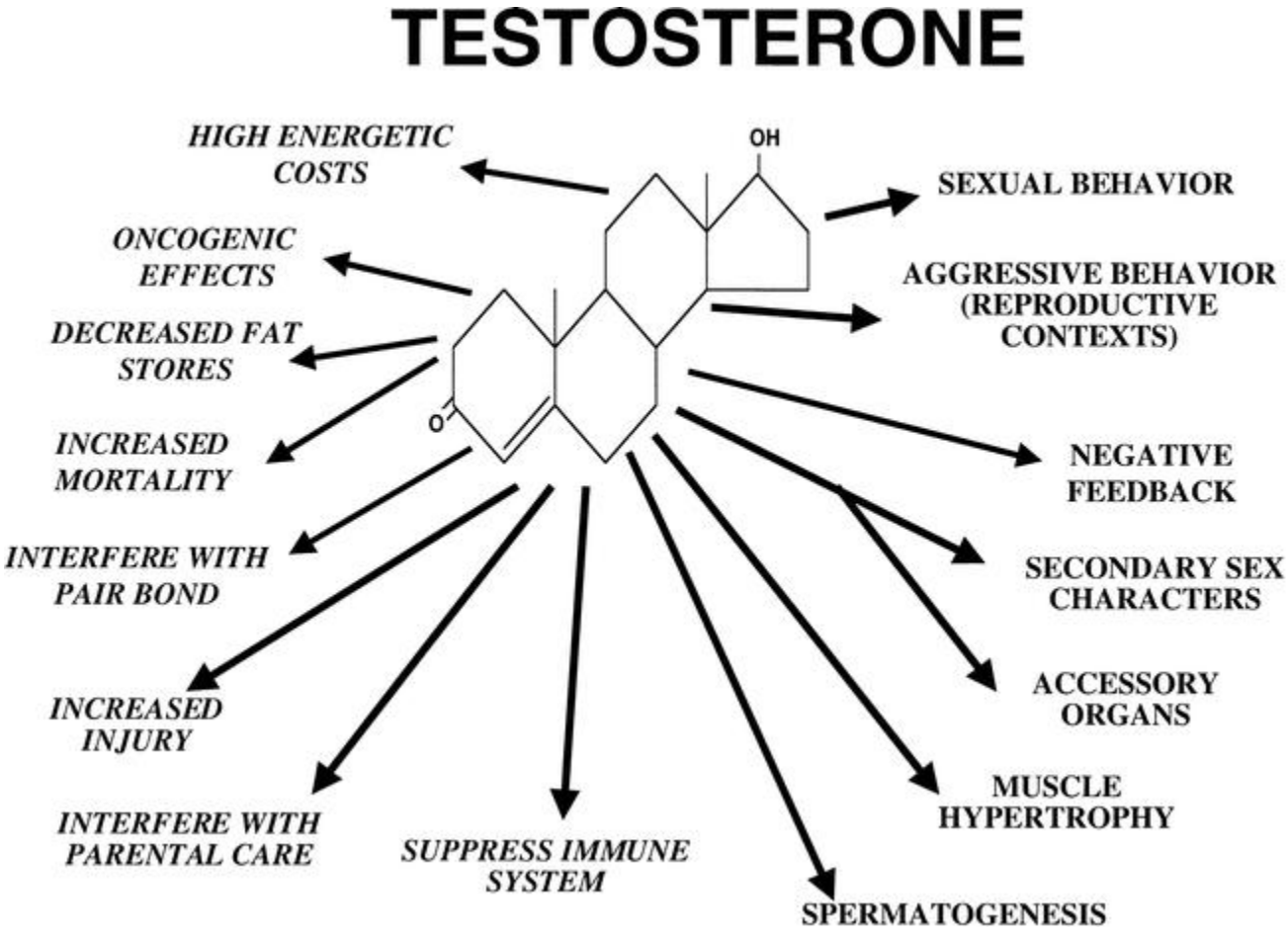
Formula: C19H2802

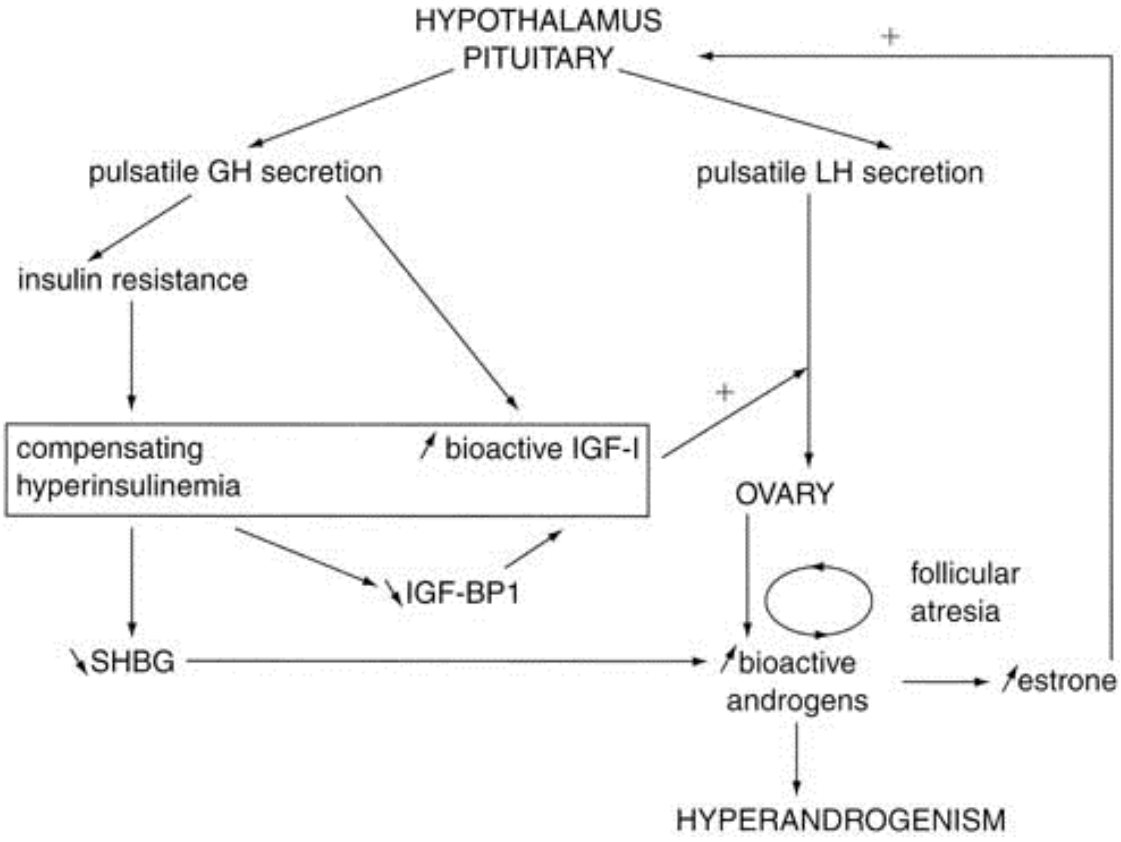
Molecular mass: 288.42 g/mol

Melting point: 155 ^{0}C

SYNTHESIS

Leydig cell is being acted upon by LH to stimulate testosterone stimulation. Feedback mechanism on hypothalamus and pituitary by testosterone and estrogen regulates the androgen synthesis.





SYNTHESIS OF TESTOSTERONE

LH + 7-transmembrane, G protein–coupled receptor

↓

cyclic AMP pathway activated

↓

LH receptor stimulated

↓

Steroid acute regulatory protein produced (rate limiting step)

↓

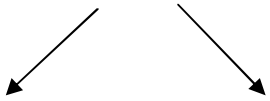
Helps delivery of cholesterol to inner mitochondrial membrane

↓

Formation of pregnenolone by CYP11A1

↓

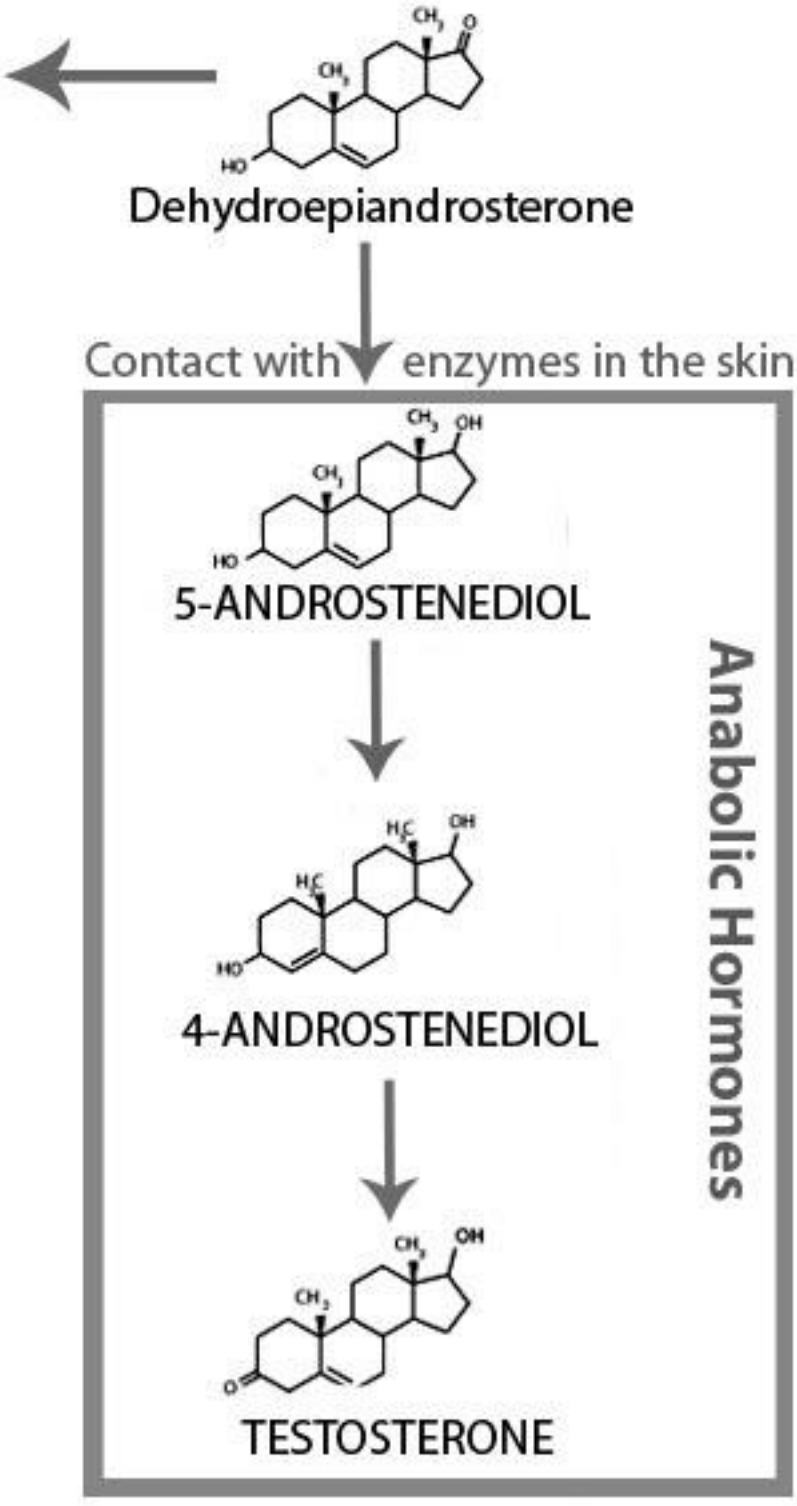
Testosterone produced



Dihydro testosterone (more potent)

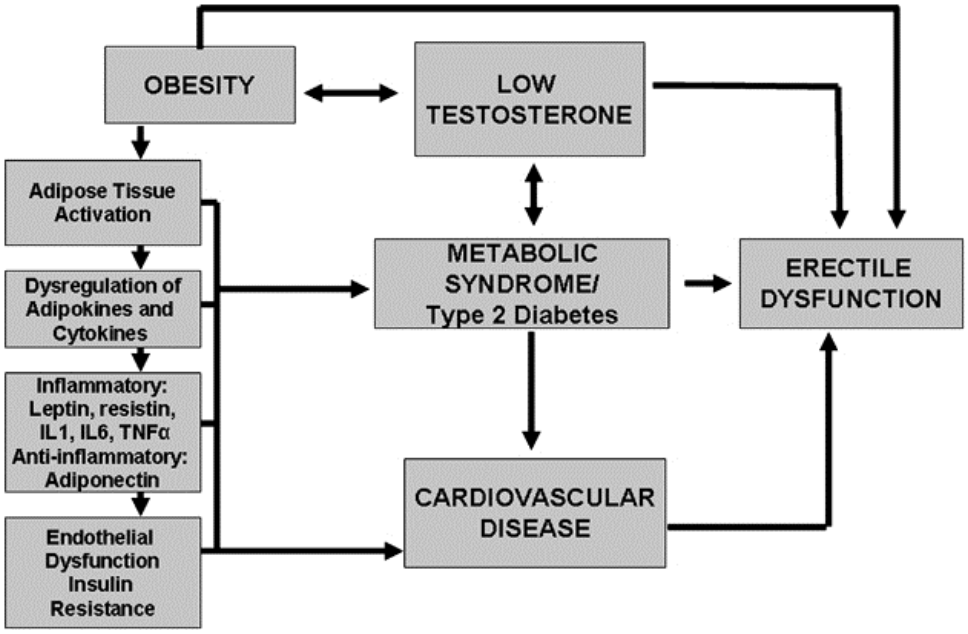
estradiol

Metabolism of the Dehydroepiandrosterone in RSTransaderm as it passes the skin and metabolizes into anabolic steroids



Testosterone has inverse relationship with visceral fat. Androgen receptors are more in number in visceral fat, as a result, it inhibits the action of lipoprotein lipase and fatty acid/triglyceride uptake. Thus limits the fat accumulation.

As the age progresses, the level of testosterone decreases and this allows increase in fat concentration. And also due to aromatization of testosterone to estradiol in obese old age patients, there is a doubt whether hormonal therapy reduces visceral obesity.



TESTOSTERONE AND CORONARY ARTERY DISEASE (CAD)

Serum testosterone has negative correlation with the extent of coronary heart disease which was proved by performing coronary angiogram. Patients who had angina and exercise induced ST segment depression have been benefited from testosterone therapy.

High androgen levels are presumed by many to explain the male predisposition to coronary artery disease. However, natural androgens inhibit male atherosclerosis.

As age advances, serum testosterone levels decrease with concurrent gonadotropin release with increased FSH and LH.

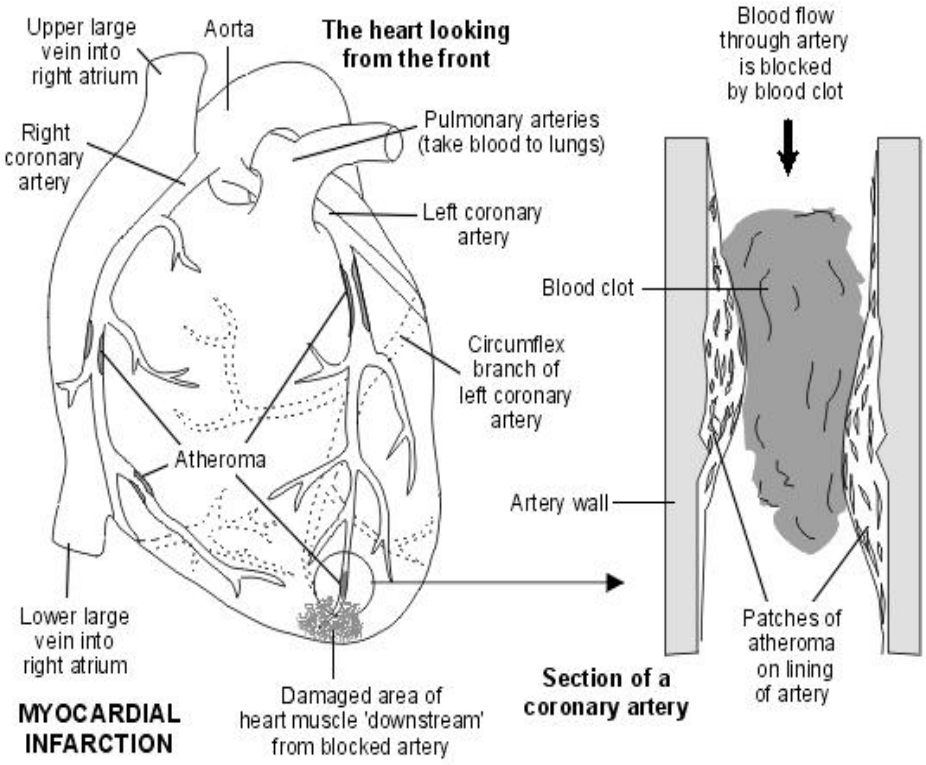
The ratio of estradiol to testosterone is raised in elderly male, which suggests that hyperestrogenemia may lead to M.I in men.

The ratio of male to female having CAD in old age group after menopause in female is 2:1. Premenopausal women are protected from endogenous estrogens while postmenopausal women are treated with exogenous estrogens. Replacement of natural analogues inhibit atheroma formation in men.

Patients who are having adverse lipid profile, raised blood pressure, obesity, insulin resistance and raised fibrinogen are found to have low testosterone levels.

Patients who are having angina symptoms and ECG changes with ischemia show improvement in testosterone therapy. Testosterone therapy has proved to have coronary and systemic vasodilatation effect in animal models in vitro.

Mortality in men is twice that of women who have protective effects of endogenous and exogenous estrogen. The abuse of anabolic steroid which resulted in sudden cardiac death leads the suggestion that it has delirious effects, but the persons who are actually having high testosterone levels have beneficial effect on cardiovascular system.



Studies have proved that intracoronary administration of testosterone have shown to increase the coronary blood flow.

Menopausal equivalent is not present in males but hormone levels decrease as the age advances, usually after the age of 30 years. Studies have proved that

males of same age group, who are having CAD have low testosterone levels when compared to men of same age group having normal coronary angiogram study.

Testosterone therapy delays the coronary event by their coronary vasodilator property.

Major function of testosterone hormone is to preserve sexual function, muscle tone, bone mineral density and post pubertal mood. Its level peaks during morning hours and also in spring, showing circadian and circannual rhythm. Testosterone is 68% bound to sex hormone binding globulin (SHBG),and 30% to albumin and remaining 2% is free form, which is biologically active.

Serum testosterone level was found to be low in established CAD patient compared to men of same age group having normal coronary angiogram.

Endogenous testosterone has direct relationship or correlation with HDL level but has indirect or negative correlation with total cholesterol, LDL and triglycerides.

Thus hypogonadal men have atherogenic dyslipidaemia whereas normal men with low testosterone have adverse lipid profile.

High testosterone levels are associated with CAD and that low serum testosterone is associated with increased aortic atheroma. Furthermore, low testosterone levels are associated with several risk factors for the development of CAD, including systemic hypertension, adverse lipid profile and high levels of fibrinogen, insulin and procoagulable factors.

Various studies shown that testosterone treatment leads to symptomatic improvement in men with angina. The acute and chronic anti-ischemic properties of testosterone mediated by coronary artery vasodilatation, which appears to involve calcium channel antagonism in a gender- specific fashion.

The androgens possess immune-modulating properties and they suppress the activity of pro-inflammatory cytokines while enhancing that of anti-inflammatory factors. Thus hypogonadal men were found to have a greater degree of inflammatory activation compared with healthy controls, including higher serum cytokine levels. Androgen therapy in these patients led to a reduction in circulating cytokines.

By the direct anti-inflammatory actions, testosterone also appears to have an important effect on rates of vascular smooth muscle cell proliferation and apoptosis which is an important factor in maintaining plaque integrity. The anabolic actions of testosterone helps in increasing protein synthesis and skeletal muscle cell size via nuclear transcription. Testosterone has been shown to inhibit human neuronal apoptosis. Importantly, it has also been shown to enhance proliferation of human vascular smooth muscle cells**.** Testosterone could, therefore, potentially be involved in maintaining the fibrous cap of the atherosclerotic plaque by promoting smooth muscle cell stability.

Men with coronary artery disease had significantly lower levels of free testosterone than did controls. The concentration of free testosterone diminished with age, and a negative correlation was found between free testosterone and body mass index. There is negative correlation between the concentration of free testosterone and lipids. A low level of free testosterone may be related to the development of coronary artery disease.

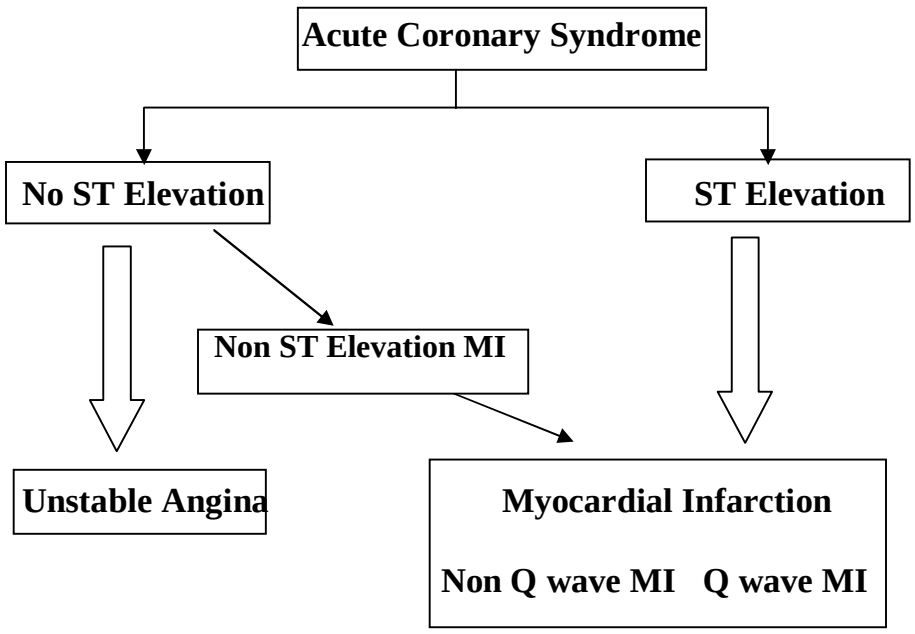
CORONARY ARTERY DISEASE ; ACUTE CORONARY SYNDROME

Coronary artery disease (CAD) remains the number one cause of death in developed countries, however, the largest projected increases in disease rates are in developing countries. Angina pectoris is the principal manifestation of coronary atherosclerosis. Patients with stable angina are at increased risk of atherosclerotic plaque instability, the development of severe myocardial ischemia and the development of acute events such as myocardial infarction (MI). In line with the changing patterns of acute events, older descriptions such as sub endocardial and Q-wave MI have now been replaced with the term acute coronary syndrome (ACS). ACS include unstable angina (UA), non-ST segment elevation myocardial infarction (NSTEMI) and ST-segment elevation myocardial infarction (STEMI).

Epidemiology

Heart disease is predominantly a disease of the elderly. The first event occurs at an average age of 65.8 years for men and 70.4 years for women. Some 50% of men and 63% of women who die suddenly from CHD have no previous symptoms of the disease. In developing countries like India, the incidence of coronary artery disease has been rising steadily. In last three decades the prevalence of coronary artery disease has increased two fold (from 2 to 4%) in rural India and three fold (from 3.45 to 9.45%) in urban India. The prevalence is higher in South India, 13% in urban areas and 7% in rural areas.

ACS patients are divided into those without and those with ST-segment elevation. The ACS without ST elevation include both UA and NSTEMI. UA is further defined as ischemic pain at rest without elevation of biomarkers, whereas NSTEMI requires increased serum biomarkers. STEMI has both ST-segment and biomarker elevation. Using these criteria. MI (either NSTEMI or STEMI) is defined as myocardial cell death due to prolonged ischemia irrespective of electrocardiogram (ECG) changes. Myocardial necrosis can be confirmed by the appearance of different proteins (biomarkers) released into the circulation by damaged myocytes. These include myoglobin, cardiac troponins T (TnT) and I (TnI), creatine kinase (CK-MB) and total creatine kinase (CK).



Pathophysiology

UA, NSTEMI and STEMI are closely related conditions with similar pathologies and clinical presentation, but with differing severity. They differ primarily in whether the ischemia is severe enough to cause sufficient myocardial damage to release detectable levels of indicators of myocardial injury, such as troponins.

The pathophysiology of atherosclerosis and ACS is well characterized. The earliest visible atherosclerotic lesion appears to be the fatty streak. Beginning in early life, these fatty streaks, or Type II lesions, are slightly raised above the intimal surface and predominantly contain foam cells derived from tissue macrophages and T lymphocytes.

Fatty steaks may progress to become mature plaques. Platelets adhere to the initial lesion and secrete growth factors including platelet-derived growth factor (PDGF) that initiate smooth muscle cell migration from the media to the intima. This is turn stimulates the production of collagen, elastin and glycoproteins, which provide the connective tissue matrix of the plaque and give it structural strength.

The lesion then becomes covered by a fibromuscular cap consisting of smooth muscle cells and collagen with a single layer of endothelial cells. This whole entity is referred to as a raised fibrolipid or advanced plaque, and becomes the substrate for plaque complications and the development of clinical symptoms.

Plaque rupture

The rupture of an atherosclerotic plaque can initiate a range of complications from UA and acute MI through to sudden cardiac death.

Acute coronary occlusion usually occurs through one of two distinct processes. First, the endothelium covering the atherosclerotic plaque can become denuded (endothelial erosion) with exposure of the prothrombotic sub endothelium to the circulating blood. Second, plaque disruption may occur with the tearing open of the fibrous cap (plaque rupture) to expose the highly thrombogenic lipid core.

The type of thrombus (platelet plug or platelet-fibrin thrombus) and the extent and duration of obstruction to coronary blood flow determine the effect on the myocardium with the development of UA, NSTEMI, or STEMI.

Plaque rupture depends more on the type of plaque than its size. Major determinants of plaque vulnerability include both local and systemic factors. Local factors include size and consistency of the atheromatous core, thickness of the fibrous cap, ongoing inflammation and repair with the cap, eccentric, plaque shape, and shear forces.

Systemic factors promoting plaque rupture include physical triggers (e.g. exertion in normally sedentary individuals), and high levels of psychological stress

with attendant increased adrenergic tone, shear forces, and generalized inflammation. The milieu of the plaque is also fundamental to its vulnerability.

Inflammation

Atherosclerosis is clearly an inflammatory disease and does not result simply from the accumulation of lipids. Low grade coronary inflammation is widespread throughout the coronary tree.

CRP, an acute phase reactant produced in the liver in response to interleukin-6 (I1-6), is both a marker and mediator of vascular inflammation as well as being a predictor of coronary events in patents with stable and unstable angina.

The most useful way of CRP testing in the emergency room setting will be among those with chest pain syndromes who have negative troponin levels.

Platelets and the coagulation system

Platelet activation is an essential step in the formation of platelet-rich thrombi. The GP IIb/IIIa receptor is responsible for platelet aggregation and is regarded as the final common pathway for platelet-platelet interaction.

Diagnosis of ACS

All chest pain patients should have a resting 12-lead ECG. Patients with chest pain can be categorized into those with

- Non-cardiac chest pain
- Stable angina
- UA
- NSTEMI or STEMI

Diagnosis is initially based on:

1. Clinical signs and symptoms
2. Resting 12-lead ECG
3. Cardiac enzymes/biomarkers.

Clinical history is important for both the diagnosis and risk stratification of patients with ACS. Age, traditional CAD risk factors, use of aspirin, and frequency of rest chest pain episodes are independent risks for adverse outcome.

Electrocardiography

A resting ECG should be obtained in all patients who present with chest pain. While the ECG is normal in about one third of chest pain patients, the pattern of ST and T wave abnormalities defines ACS patients as UA/NSTEMI versus STEMI. Severe ischemia from UA/NSTEMI may be associated with transient ST elevation, whereas the ST elevation in STEMI persists for hours. In some cases of

UA and NSTEMI the ST segments are normal, even during pain, with development of T wave inversion a few hours later. ECG findings further determine risk in ACS. In-hospital death has been reported as 8.7% with non-specific ECG changes and 11.5% with diagnostic results. Even a normal ECG carries a 5.7% 7-day mortality.

New or reversible ST-segment deviation of 0.5 mm from baseline or left bundle branch block on the admission ECG is associated with an increase in incidence of death or MI at 1 year, i.e. 15.8% versus 8.2% in patients without ECG changes. Reversible ST-segment depression is associated with a 3 to 6 fold increase in death, MI, ischemia at rest, or provocable ischemia on exercise. The ECG result and the CK level on admission can identify a difference in mortality (in the group with ST-segment elevation plus depression and elevated CK level.

Cardiac enzymes and biomarkers

Standard blood testing for the patient with suspected ACS includes TnI or TnT, total CK and CK-MB fraction. Elevated serum MB is an early marker of MI with detection as early as an hour after onset of myocardial injury.

Cardiac isoforms of TnT and TnI are more specific and sensitive indicators of myocardial damage and can detect the micro-infarctions typified by NSTEMI.

There appears little to choose between cTnT and cTnI. Studies of TnT versus TnI indicate that the two markers are equally sensitive and specific, and have similar prognostic significance.

TIMI Risk Score

- Age > 65
- ≥ 3 traditional risk factors for CAD (male sex, hyeprlipidemia, hypertension, smoking, diabetes mellitus, family history of premature CAD).
- Prior coronary stenosis > 50%.
- ST segment elevation or depression at presentation
- ≥ 2 anginal events in the prior 24 hours
- Aspirin use in the prior 7 days
- Elevated serum cardiac biomarkers.

TIMI risk score can be used to define high risk (≥ 5 points), intermediate risk (3-4 points), and low risk (0-2 points). The TIMI risk score is a validated way to assess risk of cardiac events.

Inflammatory and other prognostic markers

Inflammation has been recognized as an important feature of the pathophysiology of ACS. CRP is a strong independent marker of increased cardiovascular risk in ACS. hsCRP is also an independent predictor of risk of MI, stroke, peripheral vascular disease and sudden cardiac death even in apparently healthy individuals. Both BNP and NT-pro BNP are elevated in ACS and both are independent risk predictors of mortality.

Exercise testing

Testing should be performed in most cases within 72 hours of presentation in low or intermediate risk individuals who are free of active ischemia or heart failure symptoms for a minimum of 8-12 hours, and in intermediate risk patients after 2-3 days.

DANAMI-2 describes the prognostic importance of a pre-discharge maximal exercise test following acute MI in the era of aggressive reperfusion treatment – thrombolysis and percutaneous coronary intervention (PCI).

Echocardiography

Resting and stress echo play role in the general assessment of CAD as ischemia results in immediate changes that can be detected by echo. These include:

- Abnormalities of wall motion – hypokinetic, akinetic, dyskinetic :
- Abnormalities of wall thickening – reduced or absent systolic thickening or systolic thinning.
- Abnormalities of overall left ventricular function – e.g. ejection fraction.

Echocardiography can identify severe hypokinesis or akinesis of an infracted area, which may be helpful in patients with a non-diagnostic ECG.

Coronary angiography

Coronary angiography is regarded as the “gold standard” to define coronary anatomy. Coronary angiography should be performed in patients where the diagnosis cannot be reliably made using non-invasive testing. Patients with high-risk clinical markers, who tend to be older, have multiple risk factors and have previous MI, should undergo early coronary angiography. High-risk UA patients who are refractory to medical therapy should be referred urgently for angiography.

Management of UA/NSTEMI

General treatment guidelines

The use of combination evidence-based medical treatments including antiplatelet agents, beta-blockers, statins and ACE inhibitors has been shown to be independently and strongly associated with lower 6- month mortality in patients with ACS.

Anti-ischemic therapies

Nitrates: Their main role is to provide relief of pain. They act by arterial vasodilatation, reduced myocardial oxygen demand, reduction in coronary vasospasm, inhibition of platelet aggregation and augmentation of collateral blood flow. There is no convincing evidence that nitrates reduce rates of death or re-infarction.

Nicorandil: It is a compound with potassium-channel opener and a nitrate moiety.

The IONA study showed that nicorandil 20 mg twice daily, in addition to standard anti anginal therapy, improved outcomes in terms of reducing events related to acute coronary disease and the associated requirement for admission to hospital.

Beta-blockers

Patients with ACS with ST-segment elevation, who do not have contraindications, should be treated with beta-blockers as soon as possible to prevent acute MI/re-infarction**.** Similarly, oral beta-blockers are recommended for long-term use, indefinitely, in all patients who recover from an acute MI.

Beta-blockers reduce the odds of death in long-term trials by 23% and in short term trials by 4% following acute MI. These agents were associated with a 40% improvement in survival at 2 years, and suggested that the specific beta-blocker chosen will have little influence on mortality.

Calcium antagonists

The anti anginal effects of calcium antagonists appear to be mediated through a reduction of myocardial oxygen demand secondary to decreased afterload and myocardial contractility.

Calcium antagonists may retard the atherosclerotic process.

Oral antiplatelet therapies

Aspirin

Acute treatment with aspirin is recommended in all patients with suspected ACS in the absence of contraindications. Aspirin should be administered as soon as possible after patient presentation of suspected ACS and continued indefinitely.

Analysis of aspirin dose in the CURE study showed that medium-dose (101-199 mg/day) and high-dose ($\geq$ 200 mg/day) aspirin had no advantage over low-dose ($\leq$ 100 mg/day) aspirin whether patients received combination therapy with clopidogrel or aspirin alone.

Clopidogrel

The CURE trial compared the benefit of aspirin plus clopidogrel (300 mg loading dose, then 75 mg/day) treatment versus aspirin alone in 12,652 patients with UA or NSTEMI treated for 3-12 months.

Clopidogrel was of benefit in the CURE trial irrespective of whether high-risk features were present.

Intravenous antiplatelet therapies

GP IIb/IIIa receptor inhibitors

Several large randomized clinical trials have demonstrated improved clinical outcomes associated with the administration of GP IIb/IIIa receptor among patients presenting with ACS and after PCI.

Antithrombin therapy

Unfractionated heparin

UFH reduces thrombin generation and Xa activity. This underlies the rationale for its use in ACS.

Clinical experience

In UA, i/v UFH significantly reduced the risk of MI and recurrent refractory angina by 89% and 63% respectively.

Recommendations and dosing

A weight-based UFH regimen is preferable, with a bolus of 60 to 70 U/kg (maximum 5000 U) followed by an infusion of 12 to 15 U/kg/hour (maximum 1000 U) for 2 to 5 days.

Low molecular weight heparins

Enoxaparin is accepted as an anticoagulant in UA/NSTEMI.

Direct thrombin inhibitors

Hirudin can be used as an alternative to heparin among patients with HIT in UA/NSTEMI.

PCI ad CABG

Both FRISC-II and TACTICS-TIMI-18 demonstrated significant reduction of death, recurrent MI and hospitalization using the invasive approach. Early invasive management of UA/NSTEMI for patients with moderate to high risk.

Management of STEMI

Medical therapy

Nitrates

Oxygen

Analgesics

Morphine sulfate is the preferred analgesic agent to treat ongoing chest pain.

Antiplatelet agents

Patients should have immediate administration of non-enteric aspirin (162 mg or greater). Clopidogrel can be considered as an alternative antiplatelet agent for aspirin-sensitive patients. A 600 mg loading dose of clopidogrel may be superior. Clopidogrel enhances reperfusion with thrombolysis. Dual antiplatelet therapy reduced the composite endpoint of coronary artery occlusion, death or recurrent MI from 21.7% to 15.0%.

Infarct size limitation

- Beta-blockers

- Renin-angiotensin-aldosterone inhibition

Patients with a large anterior MI or left ventricular failure should be considered for early treatment (within first 24 hours) with an oral ACE inhibitor.

- Statin

Intensive statin therapy is effective in secondary prevention for patients presenting with ACS.

Reperfusion options for treatment of STEMI

Reperfusion: fibrinolytic agents

Fibrinolytic drug administration

Drug	Dosing	Features
Streptokinase	1.5 million units over 30-60 minutes	Lower reperfusionr ate than fibrin-specific agents, lowest cost
Alteplase	15 mg bolus - then 0.75 mg/kg over 30 minutes (maximum 50 mg) - then 0.50 mg/kg over 60 minutes (maximum 35 mg)	Lower mortality than streptokinase Short half-life requires infusion adjustment
Tenecteplase	Weight-based single bolus over 10 seconds < 60 kg = 30 mg 60-69 kg = 35 mg 70-79 kg = 40 mg 80-89 kg = 45 mg > 90kg = 50 mg	Efficacy similar to alteplase with lower bleeding complications when weight adjusted.
Reteplase	10 units over 2 minutes - then repeat 10 units at 30 minutes	Efficacy similar to alteplase

Adjunctive therapies

Antiplatelet therapy

Heparin therapy

GP IIb/IIIa inhibitors

Elective PCI after fibrinolysis

Primary PCI

Newer agents like Ranolazine

Prognosis

30% of acute MI patients and 20% with UA experience a major event (death or non-fatal coronary syndrome) during the first year after hospital admission. Some 66% of all major events during the first 6 months post-MI occur in the first 30 days. For hospitalized patients survival is determined importantly by advanced age and presence of ventricular dysfunction, with residual myocardial ischemia and cardiac arrhythmias contributing significantly. These complications highlight the importance of an aggressive early treatment strategy for UA/NSTEMI similar to STEMI patients.

MATERIALS AND METHODS

Source:

For the study, male subjects presenting with acute coronary syndrome and those patients who have recovered from a recent acute coronary event attending Medicine OPD were taken as cases. Age & sex matched healthy control were taken from the medical ward. Study was done between October 2012 and November 2013.

Number of cases - 50

Number of controls – 25

Inclusion criteria:

All male patients presenting with acute coronary syndrome between the age group of 35-74 years.

Exclusion criteria:

- Patient with liver disease
- Patient with renal parenchymal disease
- Patient with diabetes mellitus

50 male patients with acute coronary syndrome were studied based on the history, ECG changes, estimation of serum free testosterone and estimation of cardiac markers wherever required.

(a) History of angina:

Onset, situation, radiation, duration, aggravating and relieving factors were studied.

(b) ECG changes:

- Resting ECG were obtained in all patients.
- ECG studied for ST deviation, new onset left bundle branch block and significant Q waves.
-

(c) Method of serum free testosterone study:

- 50 male subjects with defined acute coronary syndrome were studied and serum free testosterone level were measured. Level of serum free testosterone was estimated by chemiluminescence Radio immuno-assay method.

Method of study

- Informed consent were obtained from all patients.
- Each patient was subjected to a detailed history and clinical examination. Clinical examination include blood pressure measurement, general physical examination, anthropometric measurement, body mass index and systemic examination.

- Biochemical parameter included fasting and random blood sugar, blood urea, serum creatinine and lipid profile.
- History of hypertension, diabetes mellitus, chronic kidney disease and dyslipidemia were noted.
- Presence of hypertension defined as per the Joint National Committee (JNC) 7 criteria.
- Presence of diabetes mellitus defined by American Diabetes Association Criteria.
- Presence of chronic kidney disease defined by National Kidney Foundation Criteria.
- Dyslipidemia as per ATP III guidelines.
- BMI calculated as per latest WHO guidelines.

Statistical analysis:

In the present study, values are expressed as mean±2SD. Variables are compared by 't' test for 2 sample mean. Attributes are compared by odds ratio and standard error of difference between two proportions by Chi-square test.

p values < 0.05 were considered significant.

OBSERVATION

In this study, 50 patients who presented with acute coronary syndrome were studied, along with 25 age and sex matched controls, who did not have any evidence of coronary artery disease.

Table - 1

Distribution of patients according to age

Age	Cases	Controls
35-44	12	10
45-54	14	11
55-64	15	4
65-74	9	0
Total	**50**	**25**

Majority of the case belonged to age group 55-64 (n=15). Mean age of the cases was 53.36 yrs.

Table - 2

Incidence of Different symptoms (n=50)

Symptoms	No. of cases	Percentage
Chest Pain	45	90%
Breathlessness	4	8%
Palpitation	28	56%
Syncope	3	6%

In the case group, most common presenting symptoms was chest pain (n=45) in 90%, followed by palpitation (n=28) in 56% breathlessness and syncope were found in 8% and 6% respectively.

Table - 3

Distribution of risk factor in cases (n=50) & controls (n=25)

Risk Factor	Cases	Controls
Hypertension	17	7
Dyslipidemia	20	4
Smoking	23	5
Alcohol	2	2
Family History	3	2

(a) Dysplipidemia is defined by any or all of the below:

T. cholesterol > 240 mg/dl

HDL-C < 40 or > 60 mg/dl

LDL-C > 130 mg/dl

Triglycerides > 160 mg/dl

Dyslipidemia was seen in 20 cases and 4 control patients.

(b) In case group (n=50), 17 patients had history of hypertension (34%) whereas in control group 7 patients had hypertension (28%)

(c) In case group, 23 (46%) were chronic smokers, and among controls groups, 5(20%) were chronic smokers.

(d) In case group, 3(6%) had family history of coronary artery disease and in control group 2(8%) had family history

Table - 4

BMI in Cases & controls

BMI	Cases	Controls
18.5-24.9	37	25
25-29.9	13	0
>30	0	0

In the case group 13 patients (26%) were over weight. In the case group, 37 patients (74%) had normal BMI while 25 patients (100%) had normal BMI in control group.

Table - 5

Mean BMI in cases and controls

Cases	Controls	p value
23.40±2.35	20.64±1.52	< 0.05 (p value = 0.000001)

BMI in cases were higher than control group and the difference was statistically highly significant.

Table - 6

Mean BP in cases and controls

BP	Cases (mmHg)	Controls (mmHg)	p value
SBP	129.36±24.79	118.8±9.27	$p < 0.05$ (p value = 0.04)
DBP	82.2±15.02	74.4±5.83	$p < 0.05$ (p value = 0.014)

Mean SBP and mean DBP were higher in cases than in controls and the difference was statistically significant.

Table - 7

Total cholesterol in case and controls

Total cholesterol (mg/dl)	Cases	Controls
< 200	19	20
201-239	8	1
> 240	23	4

In the case group, 23 patients (46%) had high levels of total cholesterol.

In the control, 4 patients (16%) had high total cholesterol.

Table - 8

Mean total cholesterol (mg/dl) in cases and controls

Cases	Controls	p value
221.4±50.10	190.2±34.05	< 0.05 (p value = 0.006)

Mean total cholesterol level was higher in cases than in control group and the difference was statistically significant.

Table - 9

HDL-C level in cases and controls

HDL-C (mg/dl)	Cases	Controls
< 40	18	4
40-60	32	21
> 60	0	0

In the case group, 18 patients (36%) had HDL-cholesterol (HDL-C) level less than 40 mg/dl and 32 (64%) had in the normal range, whereas in control group, 4 (16%) had less than 40 mg/dl and 21 (84%) subjects had in the normal range.

Table - 10

Mean HDL-C (mg/dl) in cases and controls

Cases	Controls	p value
40.34±9.91	45.16±5.96	< 0.05 (p value = 0.02)

Mean total cholesterol level was less than control group and the difference was statistically significant.

Table - 11

LDL-cholesterol level in cases and controls

HDL-C (mg/dl)	Cases	Controls
< 100	7	19
100-129	26	6
130-159	12	0
160-189	5	0
> 190	0	0

In the case group, 7 (14%) had LDL-C levels in optimal range while 19 (76%) in the control group had LDL-C levels in optimal range. 26 (52%) and 6 (24%) respectively in case and control groups had LDL-C levels in high normal range. In case group, 5 patients (10%) had high LDL-C levels while none in control group had high LDL-C level.

Table - 12

Mean LDL-C (mg/dl) in cases sand control

Cases	Controls	p value
124.14±26.0	74.94±26.39	< 0.05 (p value = 0.0001)

In the case group, LDL-C was higher than the control group and the difference was statistically significant.

Table - 13

TG level in cases and controls

HDL-C (mg/dl)	Cases	Controls
< 160	42	21
> 160	8	4

In the case group 42 (84%) had TG levels in normal range and 8 (16%) above normal.

In the control group, apparently 21(84%) had TG level in normal range.

Table - 14

Mean TG (mg/dl) level in cases and controls

Cases	Controls	p value
136.9±34.8	101.2±33.07	< 0.05 (p value = 0.00006)

The difference in TG level in case and control group was statistically significant.

Table - 15

Serum free testosterone level and coronary artery disease (ACS) cross tabulation

S. free testosterone	Cases (ACS)	Controls (without ACS)
< 9 ng/dl	40	1
> 9 ng/dl	10	24

Yates corrected Chi-square test : 35.84

Risk ratio (95% CI) : 3.32

Odds ratio (75% CI) : 96.00

p value = 0.000001 significant

In the present study group, the normal level of serum free testosterone was 9-30 ng/dl.

In the case group, 40 patients (80%) had serum free testosterone level lesser than 9 ng/dl while in control group only 1 subjects had lesser than 9 ng/dl. Serum free testosterone level was significantly decreased in patients who had acute coronary syndrome.

Table - 16

Mean serum free testosterone level (ng/dl) in cases and controls

Cases	Controls	p value
8.36±1.80	12.94±3.06	< 0.05 (p value = 0.000001)

Mean serum free testosterone level was significantly decreased in the cases than the controls and the difference was statistically significant.

Table - 17

Mean serum free testosterone level (ng/dl) cross tabulation according to number of cardiovascular risk factors in cases (n=50)

	Cases with 0-2 risk factors	**Cases with ≥ 3 risk factors**	**p value**
Mean serum free testosterone	8.02±1.23	8.58±2.08	0.33

Mean serum free testosterone level with cardiovascular risk factor did not show statistically significant difference in the present study.

Table - 18

Mean serum free testosterone (ng/dl) in hypertensive cases and hypertensive controls

Cases	Controls	p value
8.38±1.56	11.27±1.85	< 0.05 (p value = 0. 0001)

Mean serum free testosterone in hypertensive cases was found to be lesser than in hypertensive control and the study was statistically significant.

Table - 19

Mean serum free testosterone level (ng/dl) in smokers (cases and controls)

Cases	Controls	p value
8.72±1.96	11.1±2.21	< 0.05 (p value = 0. 00001)

Mean serum free testosterone in smokers cases was found to be lesser than in smoker control and the study was statistically significant.

Table No. 20

Correlation of mean serum free testosterone level with pattern of ACS

ACS	Cases	Mean serum free testosterone (ng/dl)	Percentage
AWMI	24	8.67±2.16	48%
IWMI	16	8.24±1.37	32%
UA	7	8.03±1.30	14%
AWMI+IWMI	3	7.31±1.83	6%

In the present study, most patients presented with AWMI (48%) followed by IWMI (32%).

The mean serum free testosterone level in AWMI and IWMI were 8.67±2.16 ng/dl and 8.24±1.37 ng/dl respectively.

The mean serum free testosterone level was lowest (7.31±1.83 ng/dl) in patients presented with both AWMI and IWMI.

Table No. 21

Correlation of mean serum free testosterone with mortality in acute coronary syndrome

	No. of cases	Mean Serum free testosterone (ng/dl)	Percentage
Mortality	2	10.1±4.6	4%
Survivors	48	8.28±1.66	96%

In the present study, most of the patient (96%) survived the attack while 2 patients (4%) succumbed to the illness. The mean serum free testosterone in survivors and death were 8.28±1.66 ng/dl and 10.1±4.6 ng/dl respectively . There was no correlation found between the serum free testosterone and mortality in patients with acute coronary syndrome.

DISCUSSION

Coronary artery disease (CAD) is a major cause of mortality and morbidity in developed world, and in developing countries, the incidence is rising. Atherosclerosis is the major cause of CAD. CAD manifests as acute coronary syndrome (ACS) and stable angina.

Men are more than twice as likely as women to die from coronary heart disease, and this ratio is consistent in all population and is not related to differences in risk factors. Low testosterone level are associated with CAD and low serum testosterone is associated with increased aortic atheroma.

Furthermore, low testosterone levels are associated with several risk factors for the development of CAD including systolic and diastolic hypertension, adverse lipid profile, and high levels of fibrinogen and procoagulable factors.

The present study is undertaken in order to correlate the role of serum free testosterone in patients with ACS.

Age

The mean age was 53.36±10.57 yrs in the present study group. Majority of the cases were in the age groups of 55-64 yrs (30%).

Clinical presentation

In the present study, most common presenting symptoms was chest pain (90%) followed by palpitation (56%).

Hypertension

In the present study, 34% patients had hypertension while only 28% of controls had hypertension.

The mean systolic blood pressure (SBP) in cases was 129.36±24.79 mmHg and mean diastolic blood pressure (DBP) was 82.2±15.02 mmHg.

Various other studies have shown the following parameters:

In 1994, the mean SBP was 134±22 mmHg and mean DBP was 74±14 mmHg.

In 2000 study, hypertension was found in 40.5% of cases.

In 2003 study, hypertension was found in 21.5% of cases.

In 2007 study, hypertension was found in 43.4% of cases.

The mean SBP was comparable to that seen in 2000 study was 131±7 mmHg.

The mean DBP was higher in our study group as compared to 1994 study (74±14 mmHg) and 2000 study (78±2 mmHg).

Smoking status

In the present study, 46% of cases and 20% of control were smokers.

In 2003 study, the incidence of smoking was 19%.

In 2007 study, the incidence of smoking was 37.2%.

Smoking incidence was comparatively higher in our study group, and is in accordance with community trends.

Body Mass Index (BMI)

The mean BMI in the present study was 23.40±2.35 in cases and 20.64±1.52 in controls.

The mean BMI in 2007 study was 26.2±3.1 which is higher compared to our study. The mean BMI in 2003 study was 25.5±3 and comparable to our study. The mean BMI in 2000 study was 27.7±0.7.

In 2003 study, there was a negative correlation between free testosterone and body mass index.

In 2005 study, it was found that the testosterone levels showed highly significant negative correlation with BMI (p value < 0.001).

Lipid profile

In the present study, the mean total cholesterol in cases was 221.4±50.10 mg/dl and in controls 190.2±34.05 mg/dl. The total cholesterol was higher in cases than controls and the difference was statistically significant.

In 2007 study, total cholesterol was 247±25 mg/dl.

In 2003 study, total cholesterol was 226.6±1.11 mg/dl and is comparable to our study.

The result of the present study and previous studies in accordance with cardiovascular risk attributed to total cholesterol.

HDL CHOLESTEROL:

The mean HDL cholesterol level in cases was 40.34±9.91 mg/dl and control was 45.16±5.96 mg/dl.

The mean HDL-C level in 2003 study was 51.81±0.33 mg/dl.

The mean HDL-C level in 2007 study was 47.95±0.32 mg/dl.

The lower level of HDL-C in case and control in our study is in accordance to the prevalent lipid status of South Asian population.

LDL CHOLESTEROL:

The mean LDL-cholesterol level in the present study was 124.14±26 mg/dl in cases and 74.94±26.39 mg/dl in controls, and the difference was statistically significant.

In 2007 study, the mean LDL-C level in case was 156±23 mg/dl.

In 2003 study, the mean LDL-C level was 150.42±0.98 mg/dl.

TRIGLYCERIDES:

The mean triglyceride level in the present case was 136.9±34.8 mg/dl.

The mean TG level in 2000 study was 76.95±1.09 mg/dl.

In 2003 study, the mean TG level in cases was 52.97±0.75 mg/dl.

The mean TG level in the present study was comparatively higher.

Serum free testosterone (FT)

In the present study, serum free testosterone in patients with ACS was studied and compared with controls who had cardiovascular risk factors but no evidence of CAD.

Of the 50 cases, 40 patients had serum free testosterone < 9 ng/dl which was taken as significant based on other studies done. Among 25 controls, only 1 had serum free testosterone < 9 ng/dl and 24 had serum free testosterone > 9 ng/dl.

On applying chi-square test, the odd's ratio (95% CI) is 96 and risk ratio (95% CI) is 3.32 and p value is highly significant ($p<0.001$). The means that serum free testosterone was significantly decreased in patients who had ACS.

The mean serum free testosterone in cases was 8.36±1.80 ng/dl and control was 12.94±3.06 ng/dl. The difference was statistically significant (p value < 0.001).

In 2003 study, men with coronary artery disease had significantly lower levels of free testosterone than did controls.

In 1994 study, it was found out that free testosterone correlated negatively with the degree of coronary artery disease.

In 2007 study, it was found out that the patients had significantly lower levels of testosterone than controls (9.8±6.5 mmol/l, $p<0.01$).

In 2000 study, it was concluded that men with coronary artery disease had significantly lower level of free testosterone 47.95±13.77 pmol (p value = 0.027).

In 2009 study, it was found that total and free testosterone levels of the patients with coronary artery disease were significantly lower than those of controls.

In the present study, we found that the mean serum free testosterone in patients with 0-2 cardiovascular risk factors was 8.02±1.23 ng/dl.

Mean serum free testosterone level in patients with $\geq$ 3 cardiovascular risk factors was 8.58±2.08 ng/dl.

The difference between these 2 subgroups of ACS patients was not statistically significant which means that the level of serum free testosterone level decreases irrespective of the number of cardiovascular risk factors.

The inverse relationship between testosterone levels and coronary atherosclerosis found in the present study suggests a possible protective role of the hormone on the progression of atherosclerosis.

Serum free testosterone and hypertension

The mean serum free testosterone level in hypertensive cases was 8.38±1.56 ng/dl and hypertensive control was 11.5±1.85. The difference was statistically significant.

In 2005 study, it was found that testosterone values were significantly lower in hypertensive cases.

In 1994 study, it was demonstrated that testosterone is lower in populations of men with hypertension than in normal men.

In 2007 study, a relationship between hypotestosteronemia and high blood pressure was demonstrated.

Serum free testosterone and smoking

The mean serum free testosterone in smokers with ACS was 8.72±1.96 ng/dl and serum free testosterone of smokers in control group was 11.1±2.21 ng/dl. The difference was statistically significant.

The 1990 study, 1993 study and 2000 study reported that smoking is a strong risk factors for atherosclerosis.

Serum free testosterone and patterns of acute coronary syndrome

In the present study, most patients had AWMI (48%) and IWMI (32%). Mean serum free testosterone was highest in AWMI (8.67±2.16 ng/dl) and lowest in AWMI+IWMI (7.31±1.83 ng/dl).

Serum free testosterone and mortality in ACS

In the present study, most of the patients (96%) survived the illness while 2 patients died due to ACS.

The 2007 study concluded that testosterone concentrations are inversely related to mortality due to cardiovascular disease.

SUMMARY

- The present study included 50 patients who presented with acute coronary syndrome and 25 patients of age matched controls without evidence of CAD.
- In the study group, the mean age of presentation was 53.3 yrs.
- The commonest presenting symptom was chest pain followed by palpitation.
- Hypertension present in 34% of cases and 28% of controls.
- Smoking incidence was 46% in cases and 20% in controls.
- Higher levels of total cholesterol, triglyceride and LDL cholesterol and lower level of HDL cholesterol were seen in cases. They significantly differ in comparison to controls.
- Of the cases studied, about 86% had STEMI and 14% had NSTEMI.
- The mean serum free testosterone was significantly lower in patients with ACS.
- Serum free testosterone level was also significantly lower in patients with hypertension and smokers.
- Mean serum free testosterone level did not relate to the number of cardiovascular risk factors.
- Most of the patients (48%) presented with AWMI followed by IWMI (32%).

- Mean serum free testosterone level was lowest in patient who had IWMI+AWMI as compared with other patterns of ACS.
- Of the 50 cases studied, 2 patients died (percentage of mortality - 4%).
- Mean serum free testosterone level was inversely related to hypertension, dyslipidemia, smokers and BMI.

CONCLUSION

- The serum free testosterone was found to be lower in patients with acute coronary syndrome as compared to controls. The difference between cases and controls was statistically significant.
- The serum free testosterone level was lowest in patients who presented with AWMI+IWMI as compared with other patterns of ACS.
- There was an inverse correlation found between serum free testosterone level and mortality and morbidity in patients with acute coronary syndrome.
- The serum free testosterone level was found to be lower in cases with traditional risk factors like smoking, hypertension, dyslipidemia and BMI.

BIBLIOGRAPHY

1. C.J. Malkin et al : Testosterone for secondary prevention in men with ischaemic heart disease? Q J Med 2003;96:521-529.
2. Shalender Basin et al : Disorders of the testes and male reproductive system. Harrison 17th Edition.
3. Majon Muller et al : Endogenous sex hormones and Cardiavascular disease in men.J Clin Endocrinol Metab. 88:5076-5086, 2003.
4. Chou TM et al : Testosterone induces dilatation of canine coronary conductance and resistance arteries *in vivo*. Circulation 94:2614-2619.
5. Nathan L, Chaudhuri G et al 2001 : Testosterone inhibits early atherogenesis by conversion to estradiol: critical role of aromatase.
6. Ramsay LE et al : Lancet 1996; 348:1251.
7. Oppenheim DS et al : Elevated serum lipids in hypogonadal men with and without hyperprolactinemia. Ann Intern Med 1989.
8. Pugh PJ et al : Acute haemodynamic effects of testosterone administration in men with heart failure. Euro Heart J 2002.
9. Maresca G et al : Measuring plasma fibrinogen to predict stroke and myocardial infarction- an update. Arterioscler Thromb Vasc Biol 1999.
10. Glueck CJ et al: Endogenous testosterone, fibrinolysis, and coronary heart disease risk in hyperlipidaemic men. J Lab Clin Med 1993.

11. Phillips GB, Pinkernell BH, Jing TY. The association of hypotestosteronemia with coronary artery disease in men. *Arterioscler Thromb.* 1994;14:701–706.

12. Hak AE et al . Low levels of androgens increase the risk of atherosclerosis in elderly men : the Rotterddam study. J Clin Endocrinol Metab 2002.

13. Hanke H, Lenz C, Hess B et al . Effect of Testosterone on plaque development and and androgen receptor expression in the arterial wall. Circulation 2001.

14. Moreno PR, Falk E et al. Macrophage infiltration in acute coronary syndrome:implications for plaque rupture. Circulation 1994.

15. George SJ. Tissue inhibitors of metalloproteinases in atherosclerosis. Curr Opin Lipidol 1998.

16. Geng YJ et al. Apoptosis of vascular smooth muscle cells induced by in vitro stimulation with interferon gamma, tumour necrosis factor alpha and interleukin-1 Beta. Arterioscler Thromb Vasc Biol 1996.

17. Gornstein RA et al. Androgens modulate interleukin-6 production by gingival fibroblasts in vitro. J Periodontal 1999

18. Khosla S, Atkinson EJ et al. Effect of estrogen versus testosterone on circulating osteoprotegerin and other cytokine levels in normal elderly men. J Clin Endocrinol Metab 2002.

19. Huang B et al. Association of Adipocity with prevalent coronary heart disease among elderly men: the Honolulu heart programme. Int J Relat Metab Disord 1997.

20. Rebuffe-Scrive M et al. Metoblism of adipose tissue in intra-abdominal depots of non-obese men and women. Metabolism 1989.

21. Marin P et al . Assimilation and mobilization of triglycerides in subcutaneous abdominal and femoral adipose tissue in vivo in men: effects of androgens. J Clin Endocrinol Metab 1995.

22. Schneider G, Kirshner A et al. Increased estrogen production in obese men. J Clin Endocrinol Metab 1979.

23. Snyder PJ et al. Effect of testosterone treatment on body composition and muscle strength in men over 65 years of age. J Clin Endocrinol Metab 1999.

24. Wang C et al. Transdermal testosterone gel improves sexual function, mood, muscle strength, and body composition parameters in hypogonadal men. J Clin Endocrinol Metab 2000.

25. Webb CM, Adamson DL, de Ziegler D, Collins P. Effect of acute testosterone on myocardial ischemia in men with coronary artery disease. *Am J Cardiol.* 1999;83:437–439.

26. Jaffe MD. Effect of testosterone cypionate on postexercise ST segment depression. *Br Heart J.* 1977;39:1217–1222.

27. Gray A et al . Age, disease, and changing sex hormone levels in middle-aged men. J Clin Endocrinol Metab 1991.

28. Nankin HR, Calkins JH. Decreased bioavailable testosterone in aging normal and impotent men. *J Clin Endocrinol Metab.* 1986;63:1418–1420.

29. Harman SM, Tsitouras PD, Costa PT. Reproductive hormones in aging men, II: basal pituitary gonadotropins and gonadotropin responses to luteinizing hormone-releasing hormone. *J Clin Endocrinol Metab.* 1982;54:547–551.

30. Alexandersen P, Haarbo J, Byrjalsen I, Lawaetz H, Christiansen C. Natural androgens inhibit male atherosclerosis: A study in castrated, cholesterol fed rabbits. Circ Res 1999; 84: 813–19.

31. British Heart Foundation Statistics Database. BHF, 1996: 21–2.

32. Kannel WB, Hjortland MC, McNamara PM, Gordon T. Menopause and the risk of cardiovascular disease. The Framingham Study. Ann Intern Med 1976; 85: 447–52.

33. Stampfer MJ, Colditz GA, Willett WC *et al*. Post menopausal estrogen therapy and cardiovascular disease. N Engl J Med 1991; 325: 756–62.

34. Heller R, Wheeler MJ, Micallef J. Relationship of HDL cholesterol with total and free testosterone and SHBG. Acta Endocrinologica 1983; 194: 253–6.

35. Phillips GB, Jing TY, Resnick LM, Barbagallo M, Laragh JH, Sealey JE. Sex hormones and haemostatic risk factors for coronary heart disease in men with hypertension. J Hypertens 1993; 11: 699–702.

36. Zumo. B, Strain GS, Miller LK *et al*. Plasma free and non sex hormone binding globulin bound testosterone are decreased in obese men in proportion to their degree of obesity. J Clin Endocrinol Metab 1990; 71: 929–31.

37. Haffner SM, Karhapaa P, Mykkanen L, Laakso M. Insulin Resistance, body fat distribution and sex hormones in men. Diabetes 1994; 43: 212–19.

38. Glueck CJ, Glueck HI, Stroop D, Spiers J, Hamer T, Tracy T. Endogenous testosterone, fibrinolysis and coronary heart disease risk in hyperlipidaemic men. J Lab Clin Med 1993; 122: 412–20.

39. Stigler LH, Tulgan J. Treatment of angina pectoris by testosterone propionate. NY State J Med 1943; 43: 1424–8.

40. Rosano GMC, Leonardo F, Pagnotta P *et al*. Acute antiischaemic e.ect of testosterone in men with coronary artery disease. Circulation 1999; 99: 1666–70.

41. Yue P, Chatterjee K, Beale C, Poole-Wilson, Collins P. Testosterone Relaxes Rabbit Coronary Arteries and aorta. Circulation 1995; 91: 1154–60.

42. Webb CM, Adamson DL, de Zeigler D, Collins P. E.ects of testosterone on coronary vasomotor regulation in men with coronary heart disease. Circulation 1999; 100: 1690–6.

43. Mendelsohn ME, Karas RH. The protective effects of estrogen on the cardiovascular system. *N Engl J Med.* 1999;340:1801–1811.

44. Sullivan ML, Martinez CM, Gennis P, et al. The cardiac toxicity of anabolic steroids. *Prog Cardiovasc Dis.* 1998;41:1–15.

45. English KM, Mandour O, Steeds RP, et al. Men with coronary artery disease have lower levels of androgens than men with normal coronary angiograms. *Eur Heart J.* 2000;21:890–894.

46. Alexandersen P, Haarbo J, Byrjalsen I, et al. Natural androgens inhibit male atherosclerosis: a study in castrated, cholesterol fed rabbits. *Circ Res.* 1999;84:813–819.

47. Lesser MA. Testosterone propionate therapy in one hundred cases of angina pectoris. *J Clin Endocrinol.* 1946;6:549–557.

48. Jaffe MD. Effect of testosterone cypionate on postexercise ST segment depression. *Br Heart J.* 1977;39:1217–1222.

49. Wu SZ, Weng XZ. Therapeutic effects of an androgenic preparation on myocardial ischemia and cardiac function in 62 elderly male coronary heart disease patients. *Chin Med J.* 1993;106:415–418.

50. Rosano GMC, Leonardo F, Pagnotta P, et al. Acute anti-ischemic effect of testosterone in men with coronary artery disease. *Circulation.* 1999;99:1666–1670.

51. Webb CM, Adamson DL, de Zeigler D, et al. Effect of acute testosterone on myocardial ischemia in men with coronary artery disease. *Am J Cardiol.* 1999;83:437–439.

52. Webb CM, McNeill JG, Hayward CS, et al. Effects of testosterone on coronary vasomotor regulation in men with coronary heart disease. *Circulation.* 1999;100:1690–1696.

53. Vermeulen A, Kaufman JM. Ageing of the Hypothalamo-Pituitary-Testicular Axis in Men. Horm Res 1995; **43**:25–8.

54. Wu FCW. Endocrine Aspects of anabolic steroids. Clin Chem 1997; **43**:1289–92.

55. English KM, Mandour O, Steeds RP, Diver MJ, Jones TH, Channer KS. Men with coronary artery disease have lower levels of androgens than men with normal coronary angiograms. Eur Heart J 2000; **21**:890–4.

56. English KM, Steeds RP, Jones TH, Diver MJ, Channer KS. Low-dose transdermal testosterone therapy improves angina threshold in men with chronic stable angina: A randomized double-blind placebo controlled study. Circulation 2000; **102**:1906–11.

57. Webb CM, McNeill JG, Hayward CS, de Zeigler D, Collins P. Effects of Testosterone on Coronary Vasomotor Regulation in men with Coronary Heart Disease. Circulation 1999; **100**:1690–6.

58. Levine Sa, Likoff WB. The therapeutic value of testosterone propionate in angina pectoris. N Engl J Med 1943; **229**:770–2.

59. Tremblay RR, Dube JY. Plasma concentrations of free and non Te-BG bound testosterone in women on oral contraceptives. Contraception 1974; **10**:599–605.

60. Dai WS, Gutai JP, Kuller LH, Falvo-Gerard L. Relation between plasma high-density lipoprotein cholesterol and sex hormone concentrations in men. Am J Cardiol 1984; **53**:1259–63.

61. Lichtenstsin MJ, Yarnell JWG, Elwood PC, Beswick AD, Sweetnam PM, Marks V, Teale D, Riad-Fahmy D. Sex hormones, insulin, lipids and prevalent ischemic heart disease. Am J of Epidemiol 1987; **126**:647–57.

62. Hamalainen E, Adlercreutz H, Ehnholm C, Puska P. Relationship of serum lipoproteins and apoproteins to sex hormones and to binding capacity of

sex hormone binding globulin in healthy Finnish men. Metabolism 1986; **35**:535–41.

63. Hromadova M, Hacik T, Malatinsky E, Riecansky I. Alterations of lipid metabolism in men with hypotestosteronemia. Horm Metab Res 1991; **32**:392–4.

64. Heller RF, Wheeler MJ, Micallef J, Miller NE, Lewis B. Relationship of high density lipoprotein with total and free testosterone and sex hormone binding globulin. Acta Endocrinol (Copenh) 1983; **104**:253–6.

65. Simon D, Charles M-A, Nahoul K, Orssaud G, Kremski J, Joubert E, Papoz L, Eschwege E. Association between plasma total testosterone and cardiovascular risk factors on healthy adult men: The Telecom Study. J Clin Endocrinol Metab 1997; **82**:682–5.

66. Haffner SM, Mykkanen l, Valdez RA, Katz MS. Relationship of sex hormones to lipids and lipoproteins in nondiabetic men. J Clin Endocrinol Metab 1993; **77**:1610–15.

67. Barrett-Connor E, Khaw KT. Endogenous sex hormones and cardiovascular disease in men. a prospective population-based study. Circulation 1988; **78**:539–45.

68. Barrett-Connor E. Lower endogenous androgen levels and dyslipidaemia in men with non-insulin-dependent diabetes mellitus. Ann Intern Med 1992; **117**:807–11.

69. Oppenheim DS, Greenspan SL, Zervas NT *et al.* Elevated serum lipids in hypogonadal men with and without hyperprolactinaemia. Ann Intern Med 1989; **111**:288–92.

70. Zmunda JM, Thompson PD, Dickenson, Bausserman LL. Testosterone decreases lipoprotein (A) in men. Am J Cardiol 1996; **77**:1244–7.

71. Sewdarsen M, Vythilingum S, Jialal I, Desai RK. Abnormalities in sex hormones are a risk factor for premature manifestation of coronary artery disease in South African Indian men. Atherosclerosis 1990; 83: 111–7.

72. Jeppesen LL, Jorgensen HS, Nakayama H, Raaschou HO, Olsen TS, Winther K. Decreased serum testosterone in men with acute ischemic stroke. Arterioscler Thromb Vasc Biol 1996; 16: 749–54.

73. Phillips GB, Castelli WP, Abbott RD, McNamara PM. Association of hyperestrogenemia and coronary heart disease in men in the Framingham cohort. Am J Med 1983; 74: 863–9.

74. Zhao SP, Li XP. The association of low plasma testosterone level with coronary artery disease in Chinese men. Int J Cardiol 1998; 63: 161–4.

75. English KM, Mandour O, Steeds RP, Diver MJ, Jones TH, Channer KS. Men with coronary artery disease have lower levels of androgens than men with normal coronary angiograms. Eur Heart J 2000; 21; 890–4.

76. Webb CM, McNeill JG, Hayward CS, de Zeigler D, Collins P. Effects of testosterone on coronary vasomotor regulation in men with coronary heart disease. Circulation 1999; 100: 1690–6.

77. Rosano GM, Leonardo F, Pagnotta P *et al*. Acute antiischemic effect of testosterone in men with coronary artery disease. Circulation 1999; 99: 1666–70.

78. Journals of American college of Cardiology 2003.

79. Sethi et al 'Ischaemic heart disease in" : Siddarth N Shah, Paulananad N. Acharya et al (eds.). API text book of medicine 7th Edition 2003; p:432.

80. Fox KAA et al; Findings from GRACE. Eur. Heart J. 2002. 323.

81. Michael J. Davies; Pathophysiology of acute coronary syndrome. Indian Heart J. 2000; Vol. 52; P: 473-479.

82. JACC 1993, Clin Card 1997 .

83. Ridker et al; CRP and LDL-C in cardiovascular events ;N. Eng. J. Med. 2002 ; 347.

84. Welch RD et al ;JAMA 2001 : 286.

85. Yeghiazarians et al; N. Eng. J. Med. 2000; 342.

86. Stone PH et al; Eur. H. J. 1999 ; 20.

87. Savonitto et al; Prognostic value of admission ECG in ACS ;JAMA 1999 : 281.

88. Bertrand et al; Eur. H.J. 2002 : 23 Task force on management of ACS.

89. Antman et al; The Timi Risk Score; JAMA 2000; 284.

90. Ferreiros et al Prognostic value of CRP in UA ;(Circulation, 1999 : 100).

91. Galvani et al NT Pro BNP in ACS. Circulation 2004; 110.

92. Valeur et al; Exercise testing in M.I.; Eur. Heart J. 2005.

93. Kaddoura et al S.; Echo made easy 2002.

94. Mukherjee et al ;Circulation 2004.; Evidence based medical therapy in ACS.

95. Impact of Nicorandil in Angina; Lancet 2002.

96. Lopez Sendon et al; Eur. Heart J. 2004 : 25.

97. Freemantle N. et al; BMJ 1999.

98. Journals of American college of cardiology 2002.

99. New England Journal of Medicine 2001.

100. Braunwald E et al ;ACC./AHA guidelines.

101. Barahat et al Lancet 1999; How age affects management of M.I.

INSTITUTIONAL ETHICAL COMMITTEE, STANLEY MEDICAL COLLEGE, CHENNAI-1

Title of the Work : Association between serum testosterone concentration and coronary heart disease

Principal Investigator : Dr.K.Sathesh Kumar

Designation : PG in MD (General Medicine)

Department : Department of General Medicine
Government Stanley Medical College,
Chennai-1

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 13.06.2013 at the Council Hall, Stanley Medical College, Chennai-1 at 2PM

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

13/9/13

MEMBER SECRETARY,
IEC, SMC, CHENNAI

PROFORMA

- NAME:
- AGE/SEX:
- OCCUPATION:
- ADDRESS WITH CONTACT NUMBER:
- OP/IP NO:
- DIAGNOSIS AT THE PRESENTATION:

HISTORY:

- H/O CHEST PAIN
- H/O BREATHLESSNESS
- H/O ORTHOPNEA
- H/O PALPITATION
- H/O SYNCOPE
- H/O HEMOPTYSIS

RELEVANT CLINICAL EXAMINATION:

- ANEMIA
- JAUNDICE
- CYANOSIS
- CLUBBING
- GYNAECOMASTIA
- PEDAL EDEMA
- SIGNIFICANT LYMPHADENOPATHY
- JUGULAR VENOUS PULSE

- BP
- PULSE RATE
- RESPIRATIORY RATE
- BMI
- TEMPERATURE

INSPECTION:

- CHEST WALL SYMMETRY
- APICAL IMPULSE POSITION
- PULSATION OVER PRECARDIUM
- CAROTID PULSATION
- DILATED VEINS

PALPATION:

- APICAL IMPULSE
- THRILLS OVER PRECARDIUM
- TRACHEAL POSITION
- PARASTERNAL HEAVE
- EPIGASTRIC PULSATION

AUSCULTATION:

- HEART SOUNDS IN ALL AUSCULTATORY AREA
- CARDIAC MURMURS, OTHER SOUNDS

OTHER SYSTEMS:

- RESPIRATORY SYSTEM, ABDOMEN AND CENTRAL NERVOUS SYSTEM

INVESTIGATIONS:

- CBC
- ECG
- RFT
- FBS, RBS
- FASTING LIPID PROFILE
- URINE ROUTINE
- CKMB
- CHEST X-RAY AND ECHOCARDIOGRAM
- SERUM TESTOSTERONE
- CRP
- TRPONIN-T

ஒப்புதல் படிவம்

திரு/திருமதி_____

_____

_____

_____

_____

_____

என்ற விலாசத்தில் வசிக்கும் நான் எனக்கு அளிக்கப்பட்ட தகவல் படிவத்தில் உள்ள விவரங்களை படித்தும் கேட்டும் புரிந்துகொண்டேன்.

இந்த ஆய்வின்போது எனக்கு இரத்த பரிசோதனைகள் மற்றும் ஸ்கேன் போன்ற இதர பரிசோதனைகள் செய்து கொள்ள சம்மதிக்கிறேன். மேலும் ஆய்வின் முடிவினை சொந்த அடையாளங்களை வெளியிடாமல் மருத்துவ ஆராய்ச்சிக்காக பயன்படுத்திக்கொள்ள சம்பதிக்கிறேன்.

நாள் : கையொப்பம்

இடம் : பெயர்:

தகவல் படிவம்

ஸ்டான்லி மருத்துவமனையில் பொது மருத்துவதுறையில் இருதய மாரடைப்பு நோயினால் அனுமதிக்கப்படும் நபர்களுக்கு மேற்கொள்ளப்படும் ஆய்வு தொடர்பான தகவல் படிவம் இது.

இந்த ஆய்வு க.சதீஷ்குமார் அவர்களால் அனுபவம் வாய்ந்த மருத்துவர்களின் உதவியோடு நடத்தப்படுகிறது.

இருதய மாரடைப்பினால் (Coronary Hear Disease) அனுமதிக்கப்படும் நோயாளிகளுக்கு இரத்த பரிசோதனை மற்றும் ஸ்கேன் பரிசோதனை (Echocardiogram) செய்து அதன்மூலம் உடலில் உள்ள Horomone க்கு இருதய மாரடைப்பு நோய்க்கும் சம்பந்தம் உள்ளதா என்று அறியப்படும்.

மேலும் இந்த பரிசோதனைகளால் நோயாளிகளுக்கு எந்தவிதமான பின் விளைவுகள் ஏற்பட வாய்ப்பில்லை. மேலும் நோயாளிகள் தங்கள் சுயவிருப்பத்துடன் முன்வந்தால் மட்டுமே இந்த ஆய்வு மேற்கொள்ளப்படும் என்பது உறுதியளிக்கப்படுகிறது.

Turnitin - Google Chrome

https://www.turnitin.com/newreport.asp?r=59.1275872128609&svr=7&lang=en_us&oid=382740850&pbd=2&ft=1

preferences

turnitin Originality Report

Processed on: 19-Dec-2013 18:53 IST
ID: 382740850
Word Count: 8246
Submitted: 2

Document Viewer

serum testosterone in coronary heart disease

By 20111058 . M.d. General Medicine SATHESH KUMAR K . KANAGASABATHI

Similarity Index	Similarity by Source	
23%	Internet Sources:	15%
	Publications:	23%
	Student Papers:	4%

include quoted include bibliography excluding matches < 12 words

mode: show highest matches together

INTRODUCTION Acute coronary syndrome includes unstable angina and non-ST elevated myocardial infarction and is defined as a spectrum of disease characterized by either: 1. New onset angina. 2. Angina at rest 3. Progression of angina of increasing frequency or severity. 4. Angina in response to lower levels of exertion. 5. STEMI Acute coronary syndrome most often represents acute atherosclerotic plaque rupture with exposure of thrombogenic sub-endothelial matrix. Coronary atheroma occurs as a result of inflammatory process.

Cellular inflammation and local inflammation in the arterial wall 1

occurs as a result of cytokines, which can further progress to cause

vascular smooth muscle apoptosis, degradation of the fibrin cap and plaque rupture. 1

Clinically patient can have unstable angina or progressed to myocardial infarction as a consequence of thrombus formation due to platelet aggregation. Cytokines have pivitol role in pathogenesis of atheroma formation. Pro- inflammatory cytokines are suppressed by testosterone which shows immune- modulating effect.

Men with low testosterone levels are at increase **risk of** developing acute **coronary** syndrome. **An anti-inflammatory effect of normal physiological levels of sex hormones may therefore, be important in athero-protection.** Cytokines **and** 17

C- reactive proteins are found to be elevated in coronary heart disease patients. Low serum testosterone levels is found in patients with coronary heart disease. Two most important activities in the pathogenesis of atheroma formation are

1 3% match ()
http://journals.endocrinology.org

2 1% match (Internet from 31-May-2010)
http://qjmed.oxfordjournals.org

3 1% match (Internet from 06-Oct-2010)
http://www.cenegenicsfoundation.org

4 1% match (publications)
"Abstracts", European Heart Journal, 2000.

5 1% match (publications)
MacIsaac, A.I., "Toward the quiescent coronary plaque", Journal of the American College of Cardiology, 199310

6 1% match (Internet from 16-Aug-2010)
http://www.hormonerestoration.com

7 1% match (Internet from 09-Mar-2012)
http://heart.bmj.com

8 1% match (publications)
Xin, H., and W. Jianan, "UP-REGULATION OF

21:35
19-12-2013

MASTER CHART - CASE

S.No.	I.P.No	Name	Age	Occupation	DOA	DOD/ Death	Presenting complaints	Personal History	General examination							Investigations					Diagnosis	Lipid Profile	RFT
									Pulse b.p.m.	B.P. mmHg	R/R	Hight c.m.	Weight kg	BMI	JVP	Temp.	ECG on admission	Serum free testosterone level (ng/dL)	Blood Sugar (mg%) FBS	Blood Sugar (mg%) RBS	Troponin			
1	12621	Kandhasamy	35	Driver	12.10.12	19.10.12	Chest Pain Palpitation x 1 day	Smoker	104	80/60	18	175	67	21.83	NR	Afebrile	ST↑ in I,II, III aVL. aVF, V1-V6	5.2	84	145	+ve	AWMI + IWMI	TC 246, TG 147 HDL 30 LDL 126 VLDL -29	Urea 32 Creat.0.90
2	12732	Gurumoorthy	50	Shopkeeper	18.10.12	24.10.12	Chest Pain Palpitation x 1 day	Smoker	76	110/70	18	170	75	25.95	NR	Afebrile	ST↑ in II, III aVF,	8.4	90	103	+ve	IWMI	TC 246, TG 130 HDL 51 LDL 113 VLDL27	Urea -28 Creat. 0.81
3	12789	Sudhakaran	65	Driver	07.02.13	16.02.13	Chest pain Palpitation x 4 hrs.	Tobacco Chewing	88	150/90	16	184	70	20.63	NR	Afebrile	ST↑ in precordial leads	7.1	92	130	+ve	AWMI	TC 178, TG 194,HDL 46, LDL 120 VLDL 26	Urea - 30 Creat. 0.52
4	12801	Gopalakrishnan	70	Driver	13.02.13	16.02.13	Chest Pain Nausea, Vomiting, Abdominal pain , Dizziness, Palpitation x 4 days	Smoker	58	160/110	18	175	70	22.86	NR	Afebrile	ST↑ in V1 V2 V3	7	96	136	+ve	AWMI	TC 252, TG 148, HDL 39, LDL 130, VLDL 38	Urea - 14 Creat. 0.3
5	12815	Muthusamy	40	Shopkeeper	10.02.13	18.02.13	Chest pain x 4 hrs.	Not Significant	74	160/110	24	180	74	22.79	NR	Afebrile	ST↑ in II, III aVF	6.8	78	102	+ve	IWMI	TC 260, TG 133, HDL 40, LDL 111, VLDL 33	Urea - 20 Creat-
6	12847	Mahendran	45	Worker	28.02.13	07.03.13	Chest pain, uneasiness x 6 hrs.	Smoker	102	120/70	18	172	66	22.26	NR	Afebrile	ST↑ in aVL, V1 to V6	9	84	96	"+ve	AWMI	TC 210, TG 142, HDL 48, LDL 112, VLDL 42	Urea -30
7	12901	Arockiasamy	50	Shopkeeper	05.03.13	12.03.13	Chest pain, Breathlessness, Palpitation x 2 days	Chronic smoker	120	200/120	18	171	70	23.89	NR	Afebrile	ST↑ in V1 to V3	14.3	76	110	+ve	ASMI	TC 265, TG 140, HDL 20, LDL 110, VLDL 24	Urea -25 Creat-0.6
8	12935	Krishnaraj	70	Businessman	28.03.13	02.04.13	Chestpain, Palpitationx 1 day	Tobacco Chewing	88	140/90	16	165	68	24.92	NR	Afebrile	ST↑ in II, III aVF	7.9	74	92	+ve	IWMI	TC 270, TG 145, HDL 40, LDL 120, VLDL 42	Urea -26 Creat-0.4
9	12967	Ramgopal	50	Shopkeeper	28.03.13	02.04.13	Chestpain, Palpitationx 1 day	Smoker	90	140/80	16	170	65	22.4	NR	Afebrile	ST↑ in II, III aVF	9.6	84	90	+ve	IWMI	TC 211, TG 130, HDL 54, LDL 126, VLDL 16	Urea -32 Creat-0.9
10	13112	Gopalsamy	50	Businessman	12.04.13	18.04.13	Chestpain x 5 hrs.	Not Significant	120	130/80	16	172	60	20.24	NR	Afebrile	ST↑ in I, aVL precordial leads	8.5	80	79	'+ve	Ext. AWM	TC 173., TG 214, HDL 40, LDL 90, VLDL 42	Urea -48 Creat-0.9
11	13215	Hariharan	59	Fisherman	12.04.13	20.04.13	Chestpain x 1 day	Not Significant	84	118/80	16	176	80	25.77	NR	Afebrile	ST↓ in I aVL V1 to V6	9.3	70	78	+ve	NST EMI	TC 290, TG 90, HDL 40, LDL 111, VLDL 38	Urea -30 Creat-0.9
12	13298	Prakash	64	Coolie	13.04.13	21.04.13	Chestpain since 5 hrs	Not Significant	60	150/80	16	180	70	21.56	NR	Afebrile	ST↑ in II, III aVF	11.2	86	91	+ve	IWMI	TC 255, TG 145, HDL 30, LDL 125, VLDL 18	Urea -32 Creat-0.5
13	13356	Balamurugan	47	Businessman	12.04.13	18.04.13	Chestpain x 1 day	Chronic smoker	88	140/100	16	177	70	22.29	NR	Afebrile	ST↑ in II, III aVF	8.45	95	107	+ve	IWMI	TC 167, TG 130, HDL 38, LDL 103, VLDL 26	Urea -33 Creat-0.7
14	13398	Ahamed	45	Driver	18.04.13	24.04.13	Chestpain x 8 days	Chronic smoker	104	80/60	16	170	68	23.48	Raised	Afebrile	ST↑ in I, II, III aVF, aVL	8.45	78	108	+ve	IWMI+ AWMI	TC 172, TG 130, HDL 47, LDL 136, VLDL 29	Urea -32 Creat-0.6
15	13421	Manoharan	45	Plumber	18.04.13	24.04.13	Chestpain x 4 hrs.	Smoker	88	120/80	15	177	70	22.29	NR	Afebrile	ST↑ in II, III aVF & RsR' in v1	8.15	87	102	+ve	IWMI+ RBB	TC 280, TG 139, HDL 43, LDL 112, VLDL 33	Urea -21 Creat-1
16	13498	Muthuraman	65	Electrician	06.04.13	11.04.13	Chestpain x 1 day	Smoker	78	140/90	16	174	66	22.8	NR	Afebrile	ST↑ in II, III aVF	9.8	84	97	+ve	IWMI	TC 237, TG 137, HDL 52, LDL 114, VLDL 27	Urea -33 Creat-0.8
17	13526	Ashokan	43	Policeman	23.04.13	26.04.13	Chestpain x 1 day	Not Significant	100	90/60	15	178	65	20.5	NR	Afebrile	Q wave in I & aVL, ST↑ inV5 V6	8.6	79	130	+ve	ALMI	TC 169, TG 80, HDL 48, LDL 105, VLDL 16	Urea -46 Creat-1.59
18	13701	Moorthy	64	Plumber	17.04.13	23.04.13	Chestpain x 1 day	Not Significant	86	120/80	18	173	80	26.67	NR	Afebrile	ST↑ in precordial leads	8.67	84	99	+ve	AWMI	TC 261, TG 147, HDL 35, LDL 126, VLDL 35	Urea -27 Creat-0.7
19	13820	Ramanujam	64	Coolie	15.04.13	Exp.	Chestpain, heaviness of chest, dizziness, Palpitation, vomiting x 1day	Not Significant	74	110/80	16	168	70	24.8	NR	Afebrile	ST↑ in I,aVL V4 to V6	13.4	80	86	+ve	ALMI	TC 300, TG 160, HDL 30, LDL 140, VLDL 46	Urea -30 Creat-0.1

S.No.	I.P.No	Name	Age	Occupation	DOA	DOD/ Death	Presenting complaints	Personal History	General examination							Investigations					Diagnosis	Lipid Profile	RFT
									Pulse b.p.m.	B.P. mmHg	R/R	Hight c.m.	Weight kg	BMI	JVP	Temp.	ECG on admission	Serum free testosterone level (ng/dL)	Blood Sugar (mg%)		Troponin			
																			FBS	RBS				
20	13917	Krishnan	75	Driver	23.04.13	Exp.	Breathlessness, chest discomfort x 1 day	Not Significant	92	110/70	18	170	60	20.8	NR	Afebrile	ST↑ in precordial leads	6.8	72	90	+ve	AWMI	TC 200, TG 130, HDL 40, LDL 156, VLDL 44	Urea -12 Creat-0.4
21	14002	Muthukumar	64	Electrician	24.04.13	28.04.13	Chestpain, palpitation x 1 day	Tobacco Chewing	100	130/80	16	174	74	25	NR	Afebrile	ST↑ in II,III aVF	8.6	86	120	+ve	IWMI	TC 262, TG 151, HDL 51, LDL 146, VLDL 45	Urea -37 Creat-0.56
22	14119	Raman	45	Fisherman	02.05.13	10.05.13	Chestpain x 1 day	Chronic smoker	90	150/90	16	180	70	21.6	NR	Afebrile	ST↓ in I aVL V1 to V6	7.2	74	103	+ve	AWMI	TC 210, TG 156, HDL 40, LDL 110, VLDL 42	Urea -45 Creat-0.81
23	14185	Govindhan	50	Plumber	02.05.13	09.05.13	Chestpain, palpitation, vomiting since 1 day	Not Significant	100	90/60	16	174	70	23.1	NR	Afebrile	ST↑ in II, III aVF	5.2	74	108	+ve	IWMI	TC 272, TG 171, HDL 50, LDL 148, VLDL 42	Urea -40 Creat-0.31
24	14325	Sundaram	40	Electrician	09.05.13	16.05.13	Chestpain, Palpitation x 2 hrs.	Smoker	80	120/70	16	178	70	22.1	NR	Afebrile	ST↑ in V1 to V6	8.2	81	90	+ve	AWMI	TC 300, TG 152, HDL 46, LDL 120, VLDL 42	Urea -36 Creat-0.4
25	14506	Suresh	58	Mill Worker	09.05.13	16.05.13	Chestpain x 1 day	Tobacco Chewing	78	170/110	16	179	62	19.4	NR	Afebrile	ST↑ in V1 to V3	7.7	72	84	+ve	ASMI	TC 287, TG 151, HDL 26, LDL 150, VLDL 40	Urea -38 Creat-0.6
26	14611	Ramkumar	65	Coolie	13.05.13	21.05.13	Chestpain, palpitation x 1 day	Not Significant	85	120/70	16	174	74	24.4	NR	Afebrile	ST↑ in I aVL II, III aVF, V1 to V6	8.3	76	98	+ve	IWMI+ AWMI	TC 171, TG 151, HDL 38, LDL 102, VLDL 30	Urea -16 Creat-0.03
27	14678	Suresh	55	Mill Worker	16.05.13	24.05.13		Not Significant	136	160/80	18	171	70	23.9	NR	Afebrile	ST↑ in II, III aVF	8.2	74	90	+ve	IWMI	TC 270, TG 200, HDL 38, LDL 140, VLDL 42	Urea -32 Creat-0.4
28	14721	Manoharan	43	Plumber	21.05.13	29.05.13	Chestpain, palpitation x 1day	Not Significant	100	110/70	16	178	80	25.2	NR	Afebrile	ST↑ in I aVL V1 to V6	7.8	79	104	+ve	AWMI	TC 147, TG 95, HDL 32, LDL 96, VLDL 19	Urea -32 Creat-1.01
29	14890	Sreeram	60	Driver	21.05.13	29.05.13	Chestpain, palpitation x 1 day	Smoker and alcoholics	70	140/90	20	168	60	21.3	NR	Afebrile	ST↑ in precordial leads	8	86	99	+ve	AWMI	TC 200, TG 130, HDL 43, LDL 127, VLDL 18	Urea -37 Creat-0.5
30	15200	Ramasamy	65	Pensioner	22.05.13	29.05.13	Chestpain, Palpitation, Dizziness x 1 day	Tobacco Chewing	90	130/90	16	172	72	24.3	NR	Afebrile	Normal ECG	8	85	106	+ve	NST EMI	TC 163, TG 75, HDL 47, LDL 130, VLDL 13	Urea -30 Creat-0.7
31	15401	Dinesh	41	Driver	22.05.13	30.05.13	Chestpain, palpitation x 1 day	Not Significant	86	130/80	16	170	62	21.5	NR	Afebrile	ST↑ in V1, V2,V3	6.7	80	96	+ve	ASMI	TC 239, TG 154, HDL 45, LDL 163, VLDL 30	Urea -21 Creat-0.7
32	15721	Gurusamy	40	Mill Worker	30.05.13	06.06.13	Chestpain, palpitation x 1 day	Smoker	62	130/80	16	158	70	28	NR	Afebrile	ST↑ in precordial leads	8.5	81	102	+ve	AWMI	TC 268, TG 182, HDL 25, LDL 142, VLDL 52	Urea -24 Creat-0.6
33	15832	Faisal	63	Driver	07.06.13	14.06.13	Chestpain, palpitation x 1 day	Not Significant	88	170/110	18	175	70	22.9	NR	Afebrile	ST↑ in precordial leads	8	82	130	+ve	AWMI	TC 208, TG 92, HDL 42, LDL 107, VLDL 18	Urea -19 Creat-0.1
34	16010	Azhagesan	40	Policeman	09.06.13	20.06.13	Chestpain, palpitation, sweating x 1 day	Not Significant	100	100/60	18	178	64	20.2	NR	Afebrile	ST↓ in I aVL V1 to V6	8.4	70	98	+ve	AWMI	TC 128, TG 62, HDL 30, LDL 75, VLDL 12	Urea -32 Creat-0.7
35	16125	Muthuraman	62	Teacher	09.06.13	16.06.13	Chestpain, palpitation, sweating, vomiting since 1 day	Not Significant	90	130/90	14	170	67	23.2	NR	Afebrile	ST↑ in II, III aVF	8.8	90	102	+ve	IWMI	TC 126, TG 56, HDL 27, LDL 87, VLDL 11	Urea -20 Creat-0.5
36	16303	Mohan	50	Teacher	13.6.13	19.06.13	Chestpain, Palpitation x ½ hrs.	Not Significant	100	120/80	16	168	76	26.9	NR	Afebrile	Normal ECG	5.9	99	102	+ve	NST EMI	TC 175, TG 129, HDL 30, LDL 119, VLDL 25	Urea -18 Creat-0.77
37	16534	Ashokan	42	Coolie	13.06.13	18.06.13	Chestpain x 1 day	Smoking	80	130/80	14	169	78	27.3	NR	Afebrile	T↓ in I aVL V3 to V6	7.2	90	108	+ve	NST EMI	TC 178, TG 133, HDL 42, LDL 117, VLDL 43	Urea -21 Creat-0.2
38	16855	Krishnaraj	74	Farmer	13.06.13	17.06.13	Chestpain, palpitation x 1 day	Not Significant	96	150/90	16	166	80	29	NR	Afebrile	ST↑ in II, III aVF	8.1	58	88	+ve	IWMI	TC 172, TG 148, HDL 52, LDL 90, VLDL 29	Urea -17 Creat-0.5
39	17290	Selvam	52	Coolie	20.06.06	26.06.13	Palpitation, sweating x 1 day	Smoking	100	160/110	14	165	74	27.2	NR	Afebrile	Normal ECG	9.2	82	98	+ve	NST EMI	TC 268, TG 133, HDL 54, LDL 188, VLDL 26	Urea -16 Creat-0.3

S.No.	I.P.No	Name	Age	Occupation	DOA	DOD/ Death	Presenting complaints	Personal History	General examination							Investigations					Diagnosis	Lipid Profile	RFT
									Pulse b.p.m.	B.P. mmHg	R/R	Hight c.m.	Weight kg	BMI	JVP	Temp.	ECG on admission	Serum free testosterone level (ng/dL)	Blood Sugar (mg%)		Troponin			
																			FBS	RBS				
40	17475	Rajkumar	40	Govt. Servant	21.06.13	28.06.13	Chestpain, palpitation x 1day	Not Significant	90	170/110	18	176	70	22.6	NR	Afebrile	ST↑ in precordial leads	8.9	74	108	+ve	AWMI	TC 153, TG 142, HDL 27, LDL 97, VLDL 28	Urea -20 Creat-0.5
41	17633	Muthusamy	40	Shopkeeper	21.06.13	28.06.13	Chestpain, x 1 day	Smoking	98	100/80	16	174	65	21.5	NR	Afebrile	ST↑ in I aVL V1 to V6	13.4	94	111	+ve	AWMI	TC 246, TG 118, HDL 41, LDL 181, VLDL 23	Urea -31 Creat-1
42	18011	Singaram	56	Coolie	21.06.13	30.06.13	Chestpain x 10 days	Not Significant	94	100/70	18	176	70	22.6	NR	Afebrile	ST↑ in II, III aVF	8.2	76	94	+ve	IWMI	TC 225, TG 163, HDL 62, LDL 160, VLDL 32	Urea -22 Creat-0.7
43	18305	Ramesh	45	Govt. Servant	23.06.13	30.06.13	Chestpain x 1 day	Smoking	76	140/80	16	170	56	19.4	NR	Afebrile	ST↑ in II, III aVF	8.6	82	113	+ve	IWMI	TC 252, TG 110, HDL 42, LDL 152, VLDL 13	Urea -22 Creat-0.6
44	18567	Kalimuthu	55	Farmer	26.06.13	04.07.13	Chestpain, palpitation x 1 day	Smoking	90	110/70	18	160	70	27.3	NR	Afebrile	ST↑ in II, III aVF	10.2	80	95	+ve	AWMI	TC 181, TG 120, HDL 45, LDL 123, VLDL 20	Urea -40 Creat-0.8
45	18987	Krishan	55	Govt. Servant	26.06.13	02.07.13	Chest, Palpitation, x 1 day	Smoking	106	110/70	14	174	80	26.4	NR	Afebrile	ST↑ in precordial leads	8.6	72	79	+ve	NST EMI	TC 268, TG 133, HDL 54, LDL 188, VLDL 26	Urea -16 Creat-0.3
46	19017	Muralidharan	65	Mill Worker	27.06.13	02.07.13	Breathlessness, palpitation x 1 day	Not Significant	100	110/70	16	176	71	22.9	NR	Afebrile	ST↑ in precordial leads	5.6	66	96	+ve	AWMI	TC 270, TG 130, HDL 42, LDL 100, VLDL 27	Urea -16 Creat-0.72
47	19219	Shanmugam	40	Govt. Servant	07.07.13	16.07.13	Breathlessness, palpitation x 1 day	Smoking	76	130/80	16	180	68	21	NR	Afebrile	ST↑ in V2 V3	8.8	70	84	+ve	ASMI	TC 118, TG 63, HDL 19, LDL 85, VLDL 12	Urea -28 Creat-0.79
48	19325	Ramesh	56	Shopkeeper	09.07.13	16.07.13	Chestpain x 15 days	Smoking	70	130/80	16	169	73	25.6	NR	Afebrile	ST↑ in I aVL, V2, -V6	7.3	76	85	+ve	NST EMI	TC 191, TG 215, HDL 46, LDL 101, VLDL 43	Urea -18 Creat-0.74
49	19567	Sulthan	61	Plumber	01.12.13	08.12.13	Chestpain x 1 day	Not Significant	80	140/80	16	166	60	21.8	NR	Afebrile	ST↑ in II, III aVF	6.4	84	98	+ve	IWMI	TC 270, TG 164, HDL 20, LDL 157, VLDL 36	Urea -21 Creat-0.24
50	19702	Rajan	50	Mill Worker	01.12.13	07.12.13	Chestpain x 10 days	Not Significant	84	120/70	16	174	72	23.8	NR	Afebrile	ST↑ in I, avL, V1-V6	7.4	84	95	+ve	AWMI	TC 210, TG 155, HDL 50, LDL 120, VLDL 32	Urea -25 Creat-0.94

MASTER CHART - CASE

S.No.	I.P.No	Name	Age	Occupation	DOA	DOD/ Death	Presenting complaints	Personal History	General examination								Investigations					Diagnosis	Lipid Profile	RFT
									Pulse b.p.m.	B.P. mmHg	R/R	Hight c.m.	Weight kg	BMI	JVP	Temp.	ECG on admission	Serum free testosterone level (ng/dL)	Blood Sugar (mg%)		Troponin			
																			FBS	RBS				
1	12621	Kandhasamy	35	Driver	12.10.12	19.10.12	Chest Pain Palpitation x 1 day	Smoker	104	80/60	18	175	67	21.83	NR	Afebrile	ST↑ in I,II, III aVL. aVF, V1-V6	5.2	84	145	+ve	AWMI + IWMI	TC 246, TG 147 HDL 30 LDL 126 VLDL -29	Urea 32 Creat.0.90
2	12732	Gurumoorthy	50	Shopkeeper	18.10.12	24.10.12	Chest Pain Palpitation x 1 day	Smoker	76	110/70	18	170	75	25.95	NR	Afebrile	ST↑ in II, III aVF,	8.4	90	103	+ve	IWMI	TC 246, TG 130 HDL 51 LDL 113 VLDL27	Urea -28 Creat. 0.81
3	12789	Sudhakaran	65	Driver	07.02.13	16.02.13	Chest pain Palpitation x 4 hrs.	Tobacco Chewing	88	150/90	16	184	70	20.63	NR	Afebrile	ST↑ in precordial leads	7.1	92	130	+ve	AWMI	TC 178, TG 194,HDL 46, LDL 120 VLDL 26	Urea - 30 Creat. 0.52
4	12801	Gopalakrishnan	70	Driver	13.02.13	16.02.13	Chest Pain Nausea, Vomiting, Abdominal pain , Dizziness, Palpitation x 4 days	Smoker	58	160/110	18	175	70	22.86	NR	Afebrile	ST↑ in V1 V2 V3	7	96	136	+ve	AWMI	TC 252, TG 148, HDL 39, LDL 130, VLDL 38	Urea - 14 Creat. 0.3
5	12815	Muthusamy	40	Shopkeeper	10.02.13	18.02.13	Chest pain x 4 hrs.	Not Significant	74	160/110	24	180	74	22.79	NR	Afebrile	ST↑ in II, III aVF	6.8	78	102	+ve	IWMI	TC 260, TG 133, HDL 40, LDL 111, VLDL 33	Urea - 20 Creat-
6	12847	Mahendran	45	Worker	28.02.13	07.03.13	Chest pain, uneasiness x 6 hrs.	Smoker	102	120/70	18	172	66	22.26	NR	Afebrile	ST↑ in aVL, V1 to V6	9	84	96	"+ve	AWMI	TC 210, TG 142, HDL 48, LDL 112, VLDL 42	Urea -30
7	12901	Arockiasamy	50	Shopkeeper	05.03.13	12.03.13	Chest pain, Breathlessness, Palpitation x 2 days	Chronic smoker	120	200/120	18	171	70	23.89	NR	Afebrile	ST↑ in V1 to V3	14.3	76	110	+ve	ASMI	TC 265, TG 140, HDL 20, LDL 110, VLDL 24	Urea -25 Creat-0.6
8	12935	Krishnaraj	70	Businessman	28.03.13	02.04.13	Chestpain, Palpitationx 1 day	Tobacco Chewing	88	140/90	16	165	68	24.92	NR	Afebrile	ST↑ in II, III aVF	7.9	74	92	+ve	IWMI	TC 270, TG 145, HDL 40, LDL 120, VLDL 42	Urea -26 Creat-0.4
9	12967	Ramgopal	50	Shopkeeper	28.03.13	02.04.13	Chestpain, Palpitationx 1 day	Smoker	90	140/80	16	170	65	22.4	NR	Afebrile	ST↑ in II, III aVF	9.6	84	90	+ve	IWMI	TC 211, TG 130, HDL 54, LDL 126, VLDL 16	Urea -32 Creat-0.9
10	13112	Gopalsamy	50	Businessman	12.04.13	18.04.13	Chestpain x 5 hrs.	Not Significant	120	130/80	16	172	60	20.24	NR	Afebrile	ST↑ in I, aVL precordial leads	8.5	80	79	'+ve	Ext. AWMI	TC 173., TG 214, HDL 40, LDL 90, VLDL 42	Urea -48 Creat-0.9
11	13215	Hariharan	59	Fisherman	12.04.13	20.04.13	Chestpain x 1 day	Not Significant	84	118/80	16	176	80	25.77	NR	Afebrile	ST↓ in I aVL V1 to V6	9.3	70	78	+ve	NST EMI	TC 290, TG 90, HDL 40, LDL 111, VLDL 38	Urea -30 Creat-0.9
12	13298	Prakash	64	Coolie	13.04.13	21.04.13	Chestpain since 5 hrs	Not Significant	60	150/80	16	180	70	21.56	NR	Afebrile	ST↑ in II, III aVF	11.2	86	91	+ve	IWMI	TC 255, TG 145, HDL 30, LDL 125, VLDL 18	Urea -32 Creat-0.5
13	13356	Balamurugan	47	Businessman	12.04.13	18.04.13	Chestpain x 1 day	Chronic smoker	88	140/100	16	177	70	22.29	NR	Afebrile	ST↑ in II, III aVF	8.45	95	107	+ve	IWMI	TC 167, TG 130, HDL 38, LDL 103, VLDL 26	Urea -33 Creat-0.7
14	13398	Ahamed	45	Driver	18.04.13	24.04.13	Chestpain x 8 days	Chronic smoker	104	80/60	16	170	68	23.48	Raised	Afebrile	ST↑ in I, II, III aVF, aVL	8.45	78	108	+ve	IWMI+ AWMI	TC 172, TG 130, HDL 47, LDL 136, VLDL 29	Urea -32 Creat-0.6
15	13421	Manoharan	45	Plumber	18.04.13	24.04.13	Chestpain x 4 hrs.	Smoker	88	120/80	15	177	70	22.29	NR	Afebrile	ST↑ in II, III aVF & RsR' in v1	8.15	87	102	+ve	IWMI+ RBB	TC 280, TG 139, HDL 43, LDL 112, VLDL 33	Urea -21 Creat-1
16	13498	Muthuraman	65	Electrician	06.04.13	11.04.13	Chestpain x 1 day	Smoker	78	140/90	16	174	66	22.8	NR	Afebrile	ST↑ in II, III aVF	9.8	84	97	+ve	IWMI	TC 237, TG 137, HDL 52, LDL 114, VLDL 27	Urea -33 Creat-0.8
17	13526	Ashokan	43	Policeman	23.04.13	26.04.13	Chestpain x 1 day	Not Significant	100	90/60	15	178	65	20.5	NR	Afebrile	Q wave in I & aVL, ST↑ inV5 V6	8.6	79	130	+ve	ALMI	TC 169, TG 80, HDL 48, LDL 105, VLDL 16	Urea -46 Creat-1.59
18	13701	Moorthy	64	Plumber	17.04.13	23.04.13	Chestpain x 1 day	Not Significant	86	120/80	18	173	80	26.67	NR	Afebrile	ST↑ in precordial leads	8.67	84	99	+ve	AWMI	TC 261, TG 147, HDL 35, LDL 126, VLDL 35	Urea -27 Creat-0.7
19	13820	Ramanujam	64	Coolie	15.04.13	Exp.	Chestpain, heaviness of chest, dizziness, Palpitation, vomiting x 1day	Not Significant	74	110/80	16	168	70	24.8	NR	Afebrile	ST↑ in I,aVL V4 to V6	13.4	80	86	+ve	ALMI	TC 300, TG 160, HDL 30, LDL 140, VLDL 46	Urea -30 Creat-0.1
20	13917	Krishnan	75	Driver	23.04.13	Exp.	Breathlessness, chest discomfort x 1 day	Not Significant	92	110/70	18	170	60	20.8	NR	Afebrile	ST↑ in precordial leads	6.8	72	90	+ve	AWMI	TC 200, TG 130, HDL 40, LDL 156, VLDL 44	Urea -12 Creat-0.4

S.No.	I.P.No	Name	Age	Occupation	DOA	DOD/ Death	Presenting complaints	Personal History	General examination								Investigations					Diagnosis	Lipid Profile	RFT
									Pulse b.p.m.	B.P. mmHg	R/R	Hight c.m.	Weight kg	BMI	JVP	Temp.	ECG on admission	Serum free testosterone level (ng/dL)	Blood Sugar (mg%) FBS	Blood Sugar (mg%) RBS	Troponin			
21	14002	Muthukumar	64	Electrician	24.04.13	28.04.13	Chestpain, palpitation x 1 day	Tobacco Chewing	100	130/80	16	174	74	25	NR	Afebrile	ST↑ in II,III aVF	8.6	86	120	'+ve	IWMI	TC 262, TG 151, HDL 51, LDL 146, VLDL 45	Urea -37 Creat-0.56
22	14119	Raman	45	Fisherman	02.05.13	10.05.13	Chestpain x 1 day	Chronic smoker	90	150/90	16	180	70	21.6	NR	Afebrile	ST↓ in I aVL V1 to V6	7.2	74	103	+ve	AWMI	TC 210, TG 156, HDL 40, LDL 110, VLDL 42	Urea -45 Creat-0.81
23	14185	Govindhan	50	Plumber	02.05.13	09.05.13	Chestpain, palpitation, vomiting since 1 day	Not Significant	100	90/60	16	174	70	23.1	NR	Afebrile	ST↑ in II, III aVF	5.2	74	108	+ve	IWMI	TC 272, TG 171, HDL 50, LDL 148, VLDL 42	Urea -40 Creat-0.31
24	14325	Sundaram	40	Electrician	09.05.13	16.05.13	Chestpain, Palpitation x 2 hrs.	Smoker	80	120/70	16	178	70	22.1	NR	Afebrile	ST↑ in V1 to V6	8.2	81	90	+ve	AWMI	TC 300, TG 152, HDL 46, LDL 120, VLDL 42	Urea -36 Creat-0.4
25	14506	Suresh	58	Mill Worker	09.05.13	16.05.13	Chestpain x 1 day	Tobacco Chewing	78	170/110	16	179	62	19.4	NR	Afebrile	ST↑ in V1 to V3	7.7	72	84	+ve	ASMI	TC 287, TG 151, HDL 26, LDL 150, VLDL 40	Urea -38 Creat-0.6
26	14611	Ramkumar	65	Coolie	13.05.13	21.05.13	Chestpain, palpitation x 1 day	Not Significant	85	120/70	16	174	74	24.4	NR	Afebrile	ST↑ in I aVL II, III aVF, V1 to V6	8.3	76	98	+ve	IWMI+ AWMI	TC 171, TG 151, HDL 38, LDL 102, VLDL 30	Urea -16 Creat-0.03
27	14678	Suresh	55	Mill Worker	16.05.13	24.05.13		Not Significant	136	160/80	18	171	70	23.9	NR	Afebrile	ST↑ in II, III aVF	8.2	74	90	+ve	IWMI	TC 270, TG 200, HDL 38, LDL 140, VLDL 42	Urea -32 Creat-0.4
28	14721	Manoharan	43	Plumber	21.05.13	29.05.13	Chestpain, palpitation x 1day	Not Significant	100	110/70	16	178	80	25.2	NR	Afebrile	ST↑ in I aVL V1 to V6	7.8	79	104	+ve	AWMI	TC 147, TG 95, HDL 32, LDL 96, VLDL 19	Urea -32 Creat-1.01
29	14890	Sreeram	60	Driver	21.05.13	29.05.13	Chestpain, palpitation x 1 day	Smoker and alcoholics	70	140/90	20	168	60	21.3	NR	Afebrile	ST↑ in precordial leads	8	86	99	+ve	AWMI	TC 200, TG 130, HDL 43, LDL 127, VLDL 18	Urea -37 Creat-0.5
30	15200	Ramasamy	65	Pensioner	22.05.13	29.05.13	Chestpain, Palpitation, Dizziness x 1 day	Tobacco Chewing	90	130/90	16	172	72	24.3	NR	Afebrile	Normal ECG	8	85	106	+ve	NST EMI	TC 163, TG 75, HDL 47, LDL 130, VLDL 13	Urea -30 Creat-0.7
31	15401	Dinesh	41	Driver	22.05.13	30.05.13	Chestpain, palpitation x 1 day	Not Significant	86	130/80	16	170	62	21.5	NR	Afebrile	ST↑ in V1, V2,V3	6.7	80	96	+ve	ASMI	TC 239, TG 154, HDL 45, LDL 163, VLDL 30	Urea -21 Creat-0.7
32	15721	Gurusamy	40	Mill Worker	30.05.13	06.06.13	Chestpain, palpitation x 1 day	Smoker	62	130/80	16	158	70	28	NR	Afebrile	ST↑ in precordial leads	8.5	81	102	+ve	AWMI	TC 268, TG 182, HDL 25, LDL 142, VLDL 52	Urea -24 Creat-0.6
33	15832	Faisal	63	Driver	07.06.13	14.06.13	Chestpain, palpitation x 1 day	Not Significant	88	170/110	18	175	70	22.9	NR	Afebrile	ST↑ in precordial leads	8	82	130	'+ve	AWMI	TC 208, TG 92, HDL 42, LDL 107, VLDL 18	Urea -19 Creat-0.1
34	16010	Azhagesan	40	Policeman	09.06.13	20.06.13	Chestpain, palpitation, sweating x 1 day	Not Significant	100	100/60	18	178	64	20.2	NR	Afebrile	ST↓ in I aVL V1 to V6	8.4	70	98	+ve	AWMI	TC 128, TG 62, HDL 30, LDL 75, VLDL 12	Urea -32 Creat-0.7
35	16125	Muthuraman	62	Teacher	09.06.13	16.06.13	Chestpain, palpitation, sweating, vomiting since 1 day	Not Significant	90	130/90	14	170	67	23.2	NR	Afebrile	ST↑ in II, III aVF	8.8	90	102	+ve	IWMI	TC 126, TG 56, HDL 27, LDL 87, VLDL 11	Urea -20 Creat-0.5
36	16303	Mohan	50	Teacher	13.6.13	19.06.13	Chestpain, Palpitation x ½ hrs.	Not Significant	100	120/80	16	168	76	26.9	NR	Afebrile	Normal ECG	5.9	99	102	+ve	NST EMI	TC 175, TG 129, HDL 30, LDL 119, VLDL 25	Urea -18 Creat-0.77
37	16534	Ashokan	42	Coolie	13.06.13	18.06.13	Chestpain x 1 day	Smoking	80	130/80	14	169	78	27.3	NR	Afebrile	T↓ in I aVL V3 to V6	7.2	90	108	+ve	NST EMI	TC 178, TG 133, HDL 42, LDL 117, VLDL 43	Urea -21 Creat-0.2
38	16855	Krishnaraj	74	Farmer	13.06.13	17.06.13	Chestpain, palpitation x 1 day	Not Significant	96	150/90	16	166	80	29	NR	Afebrile	ST↑ in II, III aVF	8.1	58	88	+ve	IWMI	TC 172, TG 148, HDL 52, LDL 90, VLDL 29	Urea -17 Creat-0.5
39	17290	Selvam	52	Coolie	20.06.06	26.06.13	Palpitation, sweating x 1 day	Smoking	100	160/110	14	165	74	27.2	NR	Afebrile	Normal ECG	9.2	82	98	+ve	NST EMI	TC 268, TG 133, HDL 54, LDL 188, VLDL 26	Urea -16 Creat-0.3
40	17475	Rajkumar	40	Govt. Servant	21.06.13	28.06.13	Chestpain, palpitation x 1day	Not Significant	90	170/110	18	176	70	22.6	NR	Afebrile	ST↑ in precordial leads	8.9	74	108	+ve	AWMI	TC 153, TG 142, HDL 27, LDL 97, VLDL 28	Urea -20 Creat-0.5
41	17633	Muthusamy	40	Shopkeeper	21.06.13	28.06.13	Chestpain, x 1 day	Smoking	98	100/80	16	174	65	21.5	NR	Afebrile	ST↑ in I aVL V1 to V6	13.4	94	111	+ve	AWMI	TC 246, TG 118, HDL 41, LDL 181, VLDL 23	Urea -31 Creat-1

S.No.	I.P.No	Name	Age	Occupation	DOA	DOD/ Death	Presenting complaints	Personal History	General examination								Investigations					Diagnosis	Lipid Profile	RFT
									Pulse b.p.m.	B.P. mmHg	R/R	Hight c.m.	Weight kg	BMI	JVP	Temp.	ECG on admission	Serum free testosterone level (ng/dL)	Blood Sugar (mg%) FBS	Blood Sugar (mg%) RBS	Troponin			
42	18011	Singaram	56	Coolie	21.06.13	30.06.13	Chestpain x 10 days	Not Significant	94	100/70	18	176	70	22.6	NR	Afebrile	ST↑ in II, III aVF	8.2	76	94	+ve	IWMI	TC 225, TG 163, HDL 62, LDL 160, VLDL 32	Urea -22 Creat-0.7
43	18305	Ramesh	45	Govt. Servant	23.06.13	30.06.13	Chestpain x 1 day	Smoking	76	140/80	16	170	56	19.4	NR	Afebrile	ST↑ in II, III aVF	8.6	82	113	+ve	IWMI	TC 252, TG 110, HDL 42, LDL 152, VLDL 13	Urea -22 Creat-0.6
44	18567	Kalimuthu	55	Farmer	26.06.13	04.07.13	Chestpain, palpitation x 1 day	Smoking	90	110/70	18	160	70	27.3	NR	Afebrile	ST↑ in II, III aVF	10.2	80	95	+ve	AWMI	TC 181, TG 120, HDL 45, LDL 123, VLDL 20	Urea -40 Creat-0.8
45	18987	Krishan	55	Govt. Servant	26.06.13	02.07.13	Chest, Palpitation, x 1 day	Smoking	106	110/70	14	174	80	26.4	NR	Afebrile	ST↑ in precordial leads	8.6	72	79	+ve	NST EMI	TC 268, TG 133, HDL 54, LDL 188, VLDL 26	Urea -16 Creat-0.3
46	19017	Muralidharan	65	Mill Worker	27.06.13	02.07.13	Breathlessness, palpitation x 1 day	Not Significant	100	110/70	16	176	71	22.9	NR	Afebrile	ST↑ in precordial leads	5.6	66	96	+ve	AWMI	TC 270, TG 130, HDL 42, LDL 100, VLDL 27	Urea -16 Creat-0.72
47	19219	Shanmugam	40	Govt. Servant	07.07.13	16.07.13	Breathlessness, palpitation x 1 day	Smoking	76	130/80	16	180	68	21	NR	Afebrile	ST↑ in V2 V3	8.8	70	84	+ve	ASMI	TC 118, TG 63, HDL 19, LDL 85, VLDL 12	Urea -28 Creat-0.79
48	19325	Ramesh	56	Shopkeeper	09.07.13	16.07.13	Chestpain x 15 days	Smoking	70	130/80	16	169	73	25.6	NR	Afebrile	ST↑ in I aVL, V2, -V6	7.3	76	85	+ve	NST EMI	TC 191, TG 215, HDL 46, LDL 101, VLDL 43	Urea -18 Creat-0.74
49	19567	Sulthan	61	Plumber	01.12.13	08.12.13	Chestpain x 1 day	Not Significant	80	140/80	16	166	60	21.8	NR	Afebrile	ST↑ in II, III aVF	6.4	84	98	+ve	IWMI	TC 270, TG 164, HDL 20, LDL 157, VLDL 36	Urea -21 Creat-0.24
50	19702	Rajan	50	Mill Worker	01.12.13	07.12.13	Chestpain x 10 days	Not Significant	84	120/70	16	174	72	23.8	NR	Afebrile	ST↑ in I, avL, V1-V6	7.4	84	95	+ve	AWMI	TC 210, TG 155, HDL 50, LDL 120, VLDL 32	Urea -25 Creat-0.94